



A nonlinear SAIR epidemic model: Effect of awareness class, nonlinear incidences, saturated treatment and time delay

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Abstract

Awareness plays a vital role in informing and educating people about infection risk during an outbreak and hence helps to reduce the epidemic's health burden by lowering the peak incidence. Therefore, this paper studies a susceptible-aware-infected-recovered (SAIR) epidemic model with the novel combinations of Michaelis-Menten functional type nonlinear incidence rates for unaware and aware susceptible with the inclusion of time delay as a latent period and a saturated treatment rate for infected people. The model is analyzed mathematically to describe disease transmission dynamics in two obtained equilibria: disease-free and endemic. We derive the basic reproduction number R_0 and investigate the local and global stability behavior of obtained equilibria for the time delay $\varpi \geq 0$. A bifurcation analysis is performed using center manifold theory when there is no time delay, revealing the forward bifurcation when R_0 varies from unity. Moreover, the presence of Hopf bifurcation around EE is shown depending on the bifurcation parameter time delay. Lastly, the numerical simulations validate the analytical findings.

Keywords Epidemic model with awareness · Time-delay · Michaelis-Menten incidence rates · Saturated treatment rate · Stability · Bifurcations

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1 Introduction

Epidemics remain a significant challenge for human survival. Every year, more than 8.7 million people die due to infectious diseases such as diarrhea, measles, dengue, etc. These diseases are the reason for high mortality and morbidity around the world every year. The compartmental modeling of infectious diseases is valuable in enhancing the understanding of the mechanisms of disease transmission. By analyzing the model, we can foresee future outbreaks up to a large extent which helps in evaluating control methodologies. A compartmental model describes the disease transmission mechanism by dividing the total population into distinct subclasses according to their epidemiological status. In 1927, Kermack and McKendrick [28] presented a deterministic epidemic model by considering three epidemiological compartments (classes): the susceptible compartment ($S(t)$), which measures the type of those individuals who can catch the disease and become infectives, the infective compartment ($I(t)$) that measures those individuals who are infected and can spread it to others, and the removed compartment ($R(t)$), that defines the class of those individuals who have recovered from the disease. Many epidemic models have been formulated with different compartments, such as SIS, SIR, SIRS, SEIS, SEIR, SVIRS, SFIR, etc. [8, 11, 13, 17, 20, 25, 32, 45, 50, 51].

A significant factor in the dynamic study of infectious disease is the incidence rate by which infection transmits to susceptible individuals. The incidence rate measures the frequency of occurrence of new infection per unit time. In 1927, Kermack Mckendrick assumed the bilinear incidence rate of the form $g(I)S = kIS$, where $g(I) = kI$ is unbounded for $I \geq 0$. This incidence rate is real for a small population of infected individuals, but it is unfeasible for a high density of infected populations. Therefore, numerous authors are keen to deliberate nonlinear incidence rates to study the transmission dynamics of infectious diseases (e.g., [2, 6, 19, 20, 29, 31, 36, 48, 51]). In the present article, we consider the saturating Michaelis-Menten functional response type incidence rate in which the number of adequate contacts per infective in unit time grows less rapidly as the total population increases. Michaelis-Menten contact rate is of the form $g(I) = \beta\sigma I/(1 + I)$. It combines the bilinear and standard incidence rates methods, by assuming that if the number of infectives I is suitably low, the number of actual per capita infectives $g(I)$ is proportional to I , whereas, in the case of a high density of infected individuals I , there is a saturation effect which makes the number of actual infectives constant [3, 4]. Consideration of time delay in the epidemic model emphasizes that a person may not be infectious until some time after becoming infected, which has a significant role in determining the number of infected individuals during an outbreak. Also, the inclusion of time delay in the epidemic models can induce oscillations and periodic solutions as the delay increases. Therefore, many authors focused on considering the time delay in the epidemic [24, 25, 41, 50]. Motivated by the work [3–5], in the present article, we incorporate the incidence rate of Michaelis-Menten type functional response for both susceptible and aware classes individuals with time delay representing latent period.

Affordable and safe medical treatments are also necessary, which reduce and prevent the increase in the number of infected individuals. Treatment is a powerful method that fights against the infection by stopping it from reproducing or killing bacteria.

Wang and Ruan considered a constant treatment rate in the SIR epidemic model [47]. However, a constant treatment rate is appropriate when humans have rich and better treatment resources. Therapeutic amenities and subsequent treatments might need some time to develop and implement; therefore, the choice of a reasonable treatment rate is crucial. Due to the limited medical resources, providing treatment to all the infectives puts a tremendous burden on public health associations. Hence, the saturating treatment rate, which tends to a finite limit as the number of infected individuals increases, makes the epidemic model more realistic. Therefore, Wang [46] considered a SIR epidemic model with a piecewise function type treatment rate given by

$$h(I) = \begin{cases} rI, & 0 < I \leq I_0 \\ rI_0, & I > I_0 \end{cases}$$

In this form, the treatment rate and the number of infected individuals are proportional if the maximal treatment capacity is not reached. Later, Dubey et al. deliberated different nonlinear treatment rates such as Holling Type II, Holling Type III, and Holling Type IV and studied the disease dynamics [11, 13, 14]. In the present study, for a more realistic situation of treatment availability of infected individuals, we employ a saturated functional type treatment rate given by

$$h(I) = \frac{aI^2}{bI^2 + cI + 1},$$

where, a is the maximum cure rate of infected individuals, b is the rate of limitation in treatment availability and c is the saturation constant in the absence of inhibitory effect.

Awareness about the spread of a disease is a valuable ally in affecting susceptibles' behavior and mitigating further infection. Awareness leads to sharing necessary information about the condition to the general population, getting thought, making the individuals familiar with the disorder, and providing the most substantial protection against infectious diseases. During the 2003 SARS outbreak, the Chinese Southern Weekend newspaper spread the instant message, "There is a fatal flu in Guangzhou" 126 million times in Guangzhou alone, which affected people's behavior to take necessary preventive measures [42]. This figure remains a distinct difference from the nearly low number of 5,327 cases recorded in the entire China [49]. If no centralized information is available, people can be aware of infection risk through word of mouth, personal communications, and social media like Twitter, Facebook, and other online tools by which people search out and analyze prescriptions to obtain methods for fast healing. The disease spread can also be controlled by vaccination, but immunization is costly, and sometimes vaccinations are temporary, and it is difficult to vaccinate all the individuals due to various limitations. Even some fatal diseases like AIDS, Malaria, Chikungunya, Plague, and Dengue have no vaccination; only a person's awareness can prevent the spread of these diseases efficiently and effectively. For instance, the habit of mosquito nets and mosquito coils helps in preventing Dengue and Chikungunya [15, 40]. Many authors have studied the concept of awareness in their epidemic models [1, 8, 9, 37, 38]. Funk et al., Misra et al., and Dubey et al. [12, 16, 39] deliberate the

influence and significance of awareness plans on the transmission and control of the outbreak via nonlinear mathematical models. Kumar et al. [30] incorporated the alert individuals class into the SIR epidemic model and studied the effect of alertness in infectious disease transmission dynamics. Goel et al. [18] also deliberate the influence of the full and partial awareness about the diseases spread among susceptibles with Holling type II incidences and treatment rates. A multiplicity of studies involving homogeneous spread qualitative models have been developed to provide a better understanding of the complex potential provided by awareness for the containment of epidemics. For example, in 2018, Just et al. [27] introduce awareness in the epidemic model through a specific class of aware susceptibles and investigated a reactive SAIS model in which awareness is imparted via a special class of aware susceptibles. They demonstrated that in these models, even in the presence of awareness decay, a constant fraction of aware individuals may be maintained, leading to an elevation of the epidemic threshold above the basic reproduction number. In Lacitignola et al. [34] model, awareness is introduced through a system variable ruled by an assigned evolution equation. Das et al. [10] investigate the influence of social awareness spread by media in TB transmission dynamics and gave the optimal strategy for the prevalence of tuberculosis. Lacitignola et al. [33] used the Z-control approach to gain insight into the role of awareness in the management of epidemics through the SEIR epidemic model, where they considered awareness as a time-dependent variable whose dynamics are not assigned a priori and used it as an indirect control on the class of infective individuals, exploiting its potential to produce social-distancing and self-isolation among susceptibles.

Human awareness during an outbreak is a critical factor that profoundly influences the transmission pattern of infectious diseases. Therefore, in this study, we present an infectious disease transmission compartmental model comprising four subpopulations: fully susceptible, aware susceptible population, infected, and recovered subpopulations, and formulate a mathematical time-delayed epidemic model that incorporates two explicit nonlinear incidences with a latent period and a nonlinear treatment rate for the infected individuals. We have considered the Michaelis-Menten type nonlinear incidence rate that prevents the unboundedness of the infected people and a saturating treatment rate of infected people, including the limited accessibility of treatment resources. At the beginning of the infectious disease, there is a time known as the latency period, before an infected person can transmit the infection to another person. Therefore, the inclusion of the latent period in the incidence pattern makes the present model more realistic. After formulating the model, we perform a mathematical analysis that allows long-term qualitative predictions of outbreaks and the persistence of the disease. We derive the basic reproduction number and estimate how an infection can spread in a population. Using R_0 , the local and global stability behavior of disease-free and endemic equilibria is investigated, revealing the persistence or eradication of infection. The global stability behavior of both the equilibria has been proved by using Lyapunov functionals and the Lyapunov-LaSalle invariance approach. Moreover, the oscillatory and periodic solutions near-endemic equilibrium has been seen via Hopf bifurcation by considering time delay as a bifurcation parameter. The numerical experiments show the significance of the model's variables and parameters and suggest strategies that could prevent infection.

The remainder manuscript is ordered as follows: In Sect. 2, a novel time-delayed SAIR epidemic model is presented. In Sect. 3, the basic properties of the model are presented. In Sect. 4, the local stability behavior of the disease-free equilibrium and the endemic equilibrium is investigated for the time-delay $\varpi \geq 0$ by driving the basic reproduction number R_0 . The forward bifurcation for the undelayed system is shown when the R_0 varies from unity. Further, the appearance of the periodic solutions is shown near-endemic equilibrium through Hopf bifurcation. Furthermore, the global stability of both the equilibria is investigated by employing Lyapunov functionals' direct Lyapunov method. In Sect. 5, numerical simulations are presented to show the significance of the analytical findings. To conclude, a discussion of the present study is given in Sect. 6.

2 Model derivation

Let P denotes the total population and the transmission of infectious diseases involves four types of subpopulations: Susceptible individuals $X_p(t)$, Aware individuals $A_p(t)$, Infected individuals $I_p(t)$, and Recovered individuals $R_p(t)$. That is, $P = X_p(t) + A_p(t) + I_p(t) + R_p(t)$, which means that the individuals categorized in $X_p(t)$, $A_p(t)$, $I_p(t)$ and $R_p(t)$ may vary with time t and P is a fixed population. It is assumed that each subpopulation of the SAIR model is well mixed and interact homogeneously with each other [20].

Let κ denotes the constant recruitment rate of susceptibles. ξ is the awareness rate in susceptible individuals, and thus the term ξX_p enters the class $A_p(t)$. We consider Michaelis-Menten type two explicit nonlinear incidence rates with the following interpretation: the term $\Psi(X_p(t - \varpi), I_p(t - \varpi)) = \frac{\beta \sigma X_p(t - \varpi) I_p(t - \varpi)}{1 + I_p(t - \varpi)}$ represents the incidence rate when susceptible individuals catch the infection from infected individuals, and the term $\Lambda(A_p(t - \varpi), I_p(t - \varpi)) = \frac{\gamma \sigma A_p(t - \varpi) I_p(t - \varpi)}{1 + I_p(t - \varpi)}$ represents the incidence rate when aware individuals catch the infection from infectives. Here, β and γ denote the force of infection among susceptible and aware individuals, respectively, and σ denotes the average number of contact partners. We assume that $\gamma < \beta$, as the aware individuals are at a lower risk of getting infected than fully susceptible individuals. The parameter ϖ is the time delay which represents the latent phase having a fixed duration. The period from the time of infection to becoming infectious is called the latent period. During the latent period, a host may or may not show symptoms, but the host cannot infect other hosts in both cases. Thus, the latent period significantly influences the spreading dynamics of an infectious disease or epidemic. Since the aware individuals can also become infected, perhaps at a lower rate than fully susceptibles, they also have some behavioral responses and have a latency phase due to immunological reasons. Thus, the time delay ϖ is the constant latency time and represents the time taken by the fully susceptible and aware individuals, that, infected at a time t can infect other susceptible and aware individuals at time $t + \varpi$ only. The parameters ϑ , d , and θ represent the natural death rate, disease-induced death rate, and the recovery rate of infected individuals, respectively. The nonlinear term $h(I_p(t)) = \frac{aI_p^2}{bI_p^2 + cI_p + 1}$

Table 1 Symbolizations of variables and parameters

Notation	Description
P	Total constant population
$X_p(t)$	Susceptible subpopulation (full vulnerable)
$A_p(t)$	Aware subpopulation
$I_p(t)$	Infected subpopulation
$R_p(t)$	Recovered subpopulation
ϖ	Time delay
κ	Susceptibles' recruitment rate
β	Rate of transmission of susceptible class to infected class
γ	Rate of transmission of aware class to infected class
σ	Average contact partners
ξ	Awareness rate in susceptibles
ϑ	Natural mortality rate
d	Death rate due to disease
θ	Recovery rate
a	Cure rate
b	Limitations rate in treatment availability
c	Saturation constant
$\Psi(X_p(t), I_p(t))$	Michaelis-Menten incidence rate among susceptibles
$\Lambda(X_p(t), I_p(t))$	Michaelis-Menten incidence rate among aware individuals
$h(I_p(t))$	Saturated treatment rate of infected individuals

represents the saturated treatment rate of infectives, where a denotes the maximum treatment (cure) rate, b denotes the rate of limitations in treatment availability, and c denotes the saturation constant. The description of the parameters is given in Table 1 and the transition diagram of the model (1) is shown in Fig. 1.

The resulting mathematical disease-transmission and control model based on the above assumptions is presented under the following system of delay differential equations:

$$\begin{aligned}
 \frac{dX_p}{dt} &= \kappa - \vartheta X_p - \frac{\beta\sigma X_p(t-\varpi)I_p(t-\varpi)}{1+I_p(t-\varpi)} - \xi X_p, \\
 \frac{dA_p}{dt} &= \xi X_p - \vartheta A_p - \frac{\gamma\sigma A_p(t-\varpi)I_p(t-\varpi)}{1+I_p(t-\varpi)}, \\
 \frac{dI_p}{dt} &= \frac{\beta\sigma X_p(t-\varpi)I_p(t-\varpi)}{1+I_p(t-\varpi)} + \frac{\gamma\sigma A_p(t-\varpi)I_p(t-\varpi)}{1+I_p(t-\varpi)} \\
 &\quad - (\vartheta + d + \theta)I_p - \frac{aI_p^2}{bI_p^2 + cI_p + 1}, \\
 \frac{dR_p}{dt} &= \theta I_p - \vartheta R_p + \frac{aI_p^2}{bI_p^2 + cI_p + 1}.
 \end{aligned} \tag{1}$$

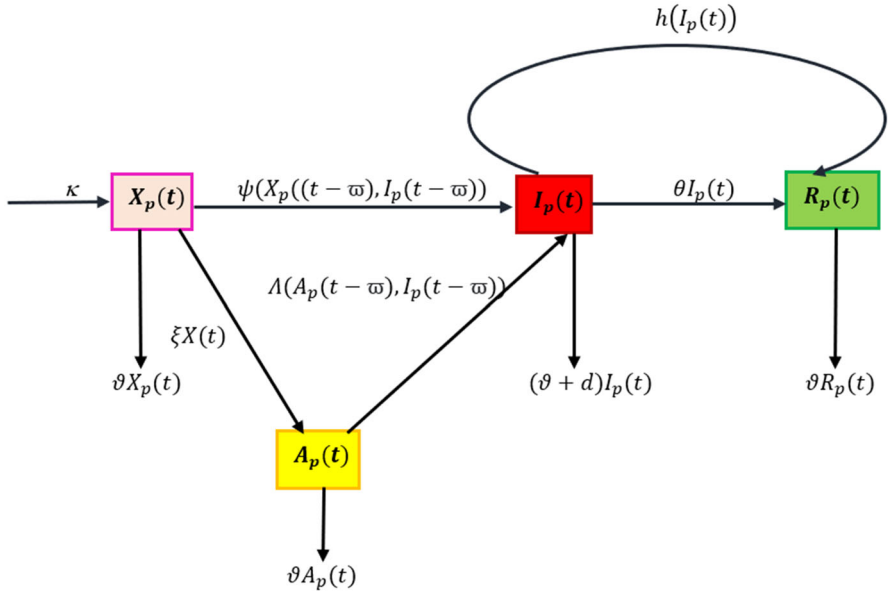


Fig. 1 Block diagram of the model (1)

subject to the initial conditions $\phi = (\phi_1, \phi_2, \phi_3, \phi_4)$ defined in the Banach space

$$C_+ = \{\phi \in C([-\varpi, 0], R_+^4) : \phi_1(\Omega) = X_p(\Omega), \phi_2(\Omega) = A_p(\Omega), \phi_3(\Omega) = I_p(\Omega), \phi_4(\Omega) = R_p(\Omega)\}, \tag{2}$$

where $R_+^4 = \{(X_p, A_p, I_p, R_p) \in R^4 : X_p \geq 0, A_p \geq 0, I_p \geq 0, R_p \geq 0\}$. Biologically, it is assumed that $\phi_i > 0$ ($i = 1, 2, 3, 4$).

We observe that the incidence functions $\Psi(X_p(t - \varpi), I_p(t - \varpi))$, $\Lambda(A_p(t - \varpi), I_p(t - \varpi))$ and the treatment rate function $h(I_p(t))$ are continuously differentiable, positive, and monotonically increasing for all $X_p(t), A_p(t), I_p(t) > 0$. That is, the following postulates hold:

- P1.** $\Psi(X_p(t), I_p(t)) > 0, \Psi'_{X_p}(X_p(t), I_p(t)) > 0, \Psi'_{I_p}(X_p(t), I_p(t)) > 0$ for $X_p(t) > 0,$
 $\Lambda(A_p(t), I_p(t)) > 0, \Lambda'_{A_p}(A_p(t), I_p(t)) > 0, \Lambda'_{I_p}(A_p(t), I_p(t)) > 0$ for $A_p(t) > 0$ and $I_p(t) > 0.$
- P2.** $\Psi(X_p(t), 0) = \Psi(0, I_p(t)) = 0, \Psi'_{X_p}(X_p(t), 0) = 0, \Psi'_{I_p}(X_p(t), 0) > 0$ for $X_p(t) > 0, I_p(t) > 0$ and,
 $\Lambda(A_p(t), 0) = \Lambda(0, I_p(t)) = 0, \Lambda'_{A_p}(A_p(t), 0) = 0, \Lambda'_{I_p}(A_p(t), 0) > 0$ for $A_p(t) > 0, I_p(t) > 0.$
- P3.** $h(0) = 0, h'(0) > 0$ for $I_p(t) \geq 0.$

3 Basic properties

Due to biological reasons, all the model's parameters κ , ϑ , β , σ , ξ , γ , d , θ , a , b , c are assumed to be positive.

Positivity: We see that the positivity of the above initial conditions for X_p , A_p , I_p and R_p in $[-\varpi, 0]$ implies positivity for all solutions $(X_p(t), A_p(t), I_p(t), R_p(t))$, $t > 0$ of model (1). We further note that $X_p(t)$ can never vanish since at each time $t > 0$ where $X_p(t)$ vanishes, it is $\frac{dX_p}{dt} = \kappa > 0$. We prove the following lemma.

Lemma 1 *The compact set*

$$D = \left\{ (X_p, A_p, I_p, R_p) \in \mathbb{R}_+^4 : X_p(t) + A_p(t) + I_p(t) + R_p(t) \leq \frac{\kappa}{\vartheta} \right\}$$

is globally attractive and invariant for the solutions of (1).

Proof The model (1) is well-posed as the right-hand side of the model (1), and its derivatives are continuous. Addition of model's equations results to

$$\begin{aligned} \frac{d}{dt}(X_p(t) + A_p(t) + I_p(t) + R_p(t)) &= \kappa - \vartheta(X_p + A_p + I_p + R_p) - dI \\ &\leq \kappa - \vartheta(X_p + A_p + I_p + R_p). \end{aligned} \quad (3)$$

Thus, we obtain

$$\begin{aligned} 0 &< X_p(t) + A_p(t) + I_p(t) + R_p(t) \\ &\leq \frac{\kappa}{\vartheta} - \left(X_p(0) + A_p(0) + I_p(0) + R_p(0) - \frac{\kappa}{\vartheta} \right) e^{-\vartheta t} \end{aligned} \quad (4)$$

Thus, the solutions of model (1) exists in the invariant region

$$0 < \lim_{t \rightarrow \infty} (X_p(t) + A_p(t) + I_p(t) + R_p(t)) \leq \frac{\kappa}{\vartheta}. \quad (5)$$

Thus, the solutions of the model (1) are closed and bounded. \square

4 Mathematical analysis

This section obtains the disease-free equilibrium (DFE), then the basic reproduction number R_0 , and investigates the system's stability at DFE. We show the existence of the endemic equilibrium (EE) and examine its stability and show the presence of Hopf bifurcation around it. Also, the global stability behavior of the DFE and EE is investigated with the help of R_0 .

We observe that the first three equations of the model (1) are free from $R(t)$; therefore, without loss of generality, we can restrict our analysis to the following reduced system

of delay differential equation:

$$\begin{aligned}
 \frac{dX_p}{dt} &= \kappa - \vartheta X_p - \frac{\beta\sigma X_p(t - \varpi)I_p(t - \varpi)}{1 + I_p(t - \varpi)} - \xi X_p, \\
 \frac{dA_p}{dt} &= \xi X_p - \vartheta A_p - \frac{\gamma\sigma A_p(t - \varpi)I_p(t - \varpi)}{1 + I_p(t - \varpi)}, \\
 \frac{dI_p}{dt} &= \frac{\beta\sigma X_p(t - \varpi)I_p(t - \varpi)}{1 + I_p(t - \varpi)} + \frac{\gamma\sigma A_p(t - \varpi)I_p(t - \varpi)}{1 + I_p(t - \varpi)} \\
 &\quad - (\vartheta + d + \theta)I_p - \frac{aI_p^2}{bI_p^2 + cI_p + 1}.
 \end{aligned} \tag{6}$$

Taking the system (6) into rest, we find that the system (6) has two equilibria:

- (i) The disease-free equilibrium (DFE): $E_0(X_0, A_0, I_0) = E_0\left(\frac{\kappa}{\vartheta + \xi}, \frac{\kappa\xi}{\vartheta(\vartheta + \xi)}, 0\right)$.
- (ii) The positive or endemic equilibrium (EE): $E_e(X_p^*, A_p^*, I_p^*)$, where X_p^*, A_p^* and I_p^* are obtained in Subsect. 4.2.

4.1 Local stability of disease-free equilibrium

The characteristic equation of the system (6) at DFE $E_0(X_0, A_0, I_0) = E_0\left(\frac{\kappa}{\vartheta + \xi}, \frac{\kappa\xi}{\vartheta(\vartheta + \xi)}, 0\right)$ is obtained as

$$(-\vartheta - \xi - \lambda)(-\vartheta - \lambda) \left(\frac{\kappa\sigma e^{-\lambda\varpi}}{\vartheta(\vartheta + \xi)} (\beta\vartheta + \xi\gamma) - (\vartheta + d + \theta + \lambda) \right) = 0. \tag{7}$$

The roots of the Eq. (7) are $\lambda_1 = -\vartheta - \xi$, $\lambda_2 = -\vartheta$, and the solutions of the transcendental equation

$$\frac{\kappa\sigma}{\vartheta(\vartheta + \xi)} (\beta\vartheta + \xi\gamma) e^{-\lambda\varpi} - (\vartheta + d + \theta) - \lambda = 0. \tag{8}$$

Assume that

$$g(\lambda) := \lambda + \vartheta + d + \theta - \frac{\kappa\sigma(\beta\vartheta + \xi\gamma)e^{-\lambda\varpi}}{\vartheta(\vartheta + \xi)} = 0. \tag{9}$$

We define the term $\frac{\kappa\sigma(\beta\vartheta + \xi\gamma)e^{-\lambda\varpi}}{\vartheta(\vartheta + \xi)(\vartheta + d + \theta)}$ at $\varpi = 0$ as the basic reproduction number R_0 of the system (6). The basic reproduction number is defined as “the average number of secondary infections caused by a single infected agent, during his/her entire infectious period, in an entirely susceptible population” [43]. Thus, the system (6) has R_0 of the form

$$R_0 = \frac{\kappa\sigma(\beta\vartheta + \xi\gamma)}{\vartheta(\vartheta + \xi)(\vartheta + d + \theta)}.$$

4.1.1 Analysis for $R_0 \neq 1$

The roots λ_1 and λ_2 of Eq. (7) preserve negative signs. Therefore, the analysis is now based on the Eq. (9). We see that

$$g(0) = \vartheta + d + \theta - \frac{\kappa\sigma(\beta\vartheta + \xi\gamma)}{\vartheta(\vartheta + \xi)} = (\vartheta + d + \theta)(1 - R_0). \quad (10)$$

If $R_0 > 1$, then $g(0) < 0$. Also,

$$g'(\lambda) = 1 + \varpi \frac{\kappa\sigma(\beta\vartheta + \xi\gamma)e^{-\lambda\varpi}}{\vartheta(\vartheta + \xi)} > 0. \quad (11)$$

Hence, $g(0) < 0$ and $g'(\lambda) > 0$ imply that when $R_0 > 1$, then the root of $g(\lambda) = 0$ is unique, real and positive.

Note that,

$$\begin{aligned} \operatorname{Re} \lambda &= \frac{\kappa\sigma(\beta\vartheta + \xi\gamma) \cos(\operatorname{Im} \lambda)\varpi}{\vartheta(\vartheta + \xi)} e^{-(\operatorname{Re} \lambda)\varpi} - (\vartheta + d + \theta) \\ &< \frac{\kappa\sigma(\beta\vartheta + \xi\gamma)}{\vartheta(\vartheta + \xi)} - (\vartheta + d + \theta) \\ &< 0, \quad \text{provided } R_0 < 1. \end{aligned} \quad (12)$$

Therefore, $R_0 < 1$ implies that Eq. (7) has a root λ with negative real part. Thus, the following theorem is stated:

Theorem 1 For $\varpi \geq 0$, the disease-free equilibrium (DFE) E_0 is

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

4.1.2 Analysis at $R_0 = 1$

Now we analyze the system (6) at E_0 and $R_0 = 1$ for $\varpi > 0$ and $\varpi = 0$, separately.

Case (i) $\varpi > 0$

When $R_0 = 1$, then Eq. (9) has a simple characteristic root $\lambda = 0$.

It is noticed that $R_0 = 1$ gives $\kappa\sigma(\beta\vartheta + \xi\gamma) = \vartheta(\vartheta + \xi)(\vartheta + d + \theta)$.

Let $\lambda = p + iq$ be the other solution of Eq. (9), then we get:

$$p + iq + d + \vartheta + \theta - (\cos q\varpi - i \sin q\varpi)(d + \vartheta + \theta)e^{-p\varpi} = 0. \quad (13)$$

Applying Euler's formula and then splitting real part and imaginary part of Eq. (13) yields

$$\begin{aligned} p + d + \vartheta + \theta &= e^{-p\varpi} (d + \vartheta + \theta) \cos q\varpi, \\ q &= -(d + \vartheta + \theta) e^{-p\varpi} \sin q\varpi. \end{aligned} \quad (14)$$

A root that satisfies both equations of (14) must be a solution to the equation attained by squaring and adding these two equations. Hence, we get

$$(p + d + \vartheta + \theta)^2 + q^2 = (d + \vartheta + \theta)^2 e^{-2p\varpi}. \tag{15}$$

For Eq. (15) to hold, we must have $p \leq 0$. Thus, E_0 is linearly neutrally stable. Thus, the following theorem is stated:

Theorem 2 *The disease-free equilibrium E_0 at $R_0 = 1$ of the system (6) is linearly neutrally stable for $\varpi > 0$.*

Case (ii) $\varpi = 0$

In this case, we study the qualitative behavior of the undelayed system (6) (i.e., $\varpi = 0$) through the stability analysis near critical points, i.e., at E_0 and $R_0 = 1$, using the bifurcation theory approach [7], depending upon the center manifold theory [21]. Here, we are keen on evaluating if there is a stable coexistence endemic equilibrium that bifurcates from E_0 , and E_0 converts its behavior from stable to unstable, which is known as forward bifurcation.

For simplicity, let $X_p = x_1$, $A_p = x_2$, and $I_p = x_3$. So, the system (6) reduces to

$$\begin{aligned} \frac{dx_1}{dt} &= \kappa - \vartheta x_1 - \frac{\beta \sigma x_1(t)x_3(t)}{1 + x_3(t)} - \xi x_1 \equiv f_1, \\ \frac{dx_2}{dt} &= \xi x_1 - \vartheta x_2 - \frac{\gamma \sigma x_2(t)x_3(t)}{1 + x_3(t)} \equiv f_2, \\ \frac{dx_3}{dt} &= \frac{\beta \sigma x_1(t)x_3(t)}{1 + x_3(t)} + \frac{\gamma \sigma x_2(t)x_3(t)}{1 + x_3(t)} - (\vartheta + d + \theta)x_3 - \frac{ax_3^2}{bx_3^2 + cx_3 + 1} \equiv f_3. \end{aligned} \tag{16}$$

Observe that $R_0 = 1 \iff$ the bifurcation parameter $\sigma = \sigma^* = \frac{\vartheta(\vartheta + \xi)(\vartheta + d + \theta)}{\kappa(\beta\vartheta + \xi\gamma)}$. The Jacobian matrix $J(E_0, \sigma^*)$ of (16) obtained at E_0 and σ^* is

$$J(E_0, \sigma^*) = \begin{bmatrix} -\vartheta - \xi & 0 & -\frac{\beta\sigma^*\kappa}{\vartheta + \xi} \\ \xi & -\vartheta & -\frac{\gamma\sigma^*\kappa\xi}{\vartheta(\vartheta + \xi)} \\ 0 & 0 & 0 \end{bmatrix}.$$

$J(E_0, \sigma^*)$ has eigenvalues $\lambda_1 = -\vartheta - \xi$, $\lambda_2 = -\vartheta$, and $\lambda_3 = 0$. Since λ_3 is a simple zero eigenvalue of $J(E_0, \sigma^*)$. Hence, when $R_0 = 1$, the DFE E_0 is a non-hyperbolic equilibrium.

The right eigenvector $v = (v_1, v_2, v_3)$ corresponding to $\lambda_3 = 0$ of the Jacobian matrix $J(E_0, \sigma^*)$ is obtained as

$$\begin{aligned}v_1 &= -\frac{\beta\kappa\sigma^*}{(\vartheta + \xi)^2}, \\v_2 &= -\frac{\kappa\vartheta\xi\sigma^*(\beta + \gamma) + \gamma\kappa\xi^2\sigma^*}{\vartheta^2(\vartheta + \xi)^2}, \\v_3 &= 1.\end{aligned}$$

The left eigenvector $w = (w_1, w_2, w_3)$ of the Jacobian matrix $J(E_0, \sigma^*)$ corresponding to $\lambda_3 = 0$ is obtained as

$$\begin{aligned}w_1 &= 0, \\w_2 &= 0, \\w_3 &= 1.\end{aligned}$$

Assume that the right-hand side of the system (16) is denoted by f_k . Then, the bifurcation coefficients a_1 and b_1 defined in Theorem 4.1 of [7] are given by:

$$\begin{aligned}a_1 &= \sum_{k,i,j=1}^3 w_k v_i v_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j} \right)_{E_0}, \\b_1 &= \sum_{k,i=1}^3 w_k v_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \sigma^*} \right)_{E_0}.\end{aligned}$$

The non-zero partial derivative of the functions f_k 's calculated at E_0 are evaluated as

$$\begin{aligned}\left(\frac{\partial^2 f_3}{\partial x_3 \partial x_1} \right)_{E_0} &= \beta\sigma^*, \quad \left(\frac{\partial^2 f_3}{\partial x_3 \partial x_2} \right)_{E_0} = \gamma\sigma^*, \quad \left(\frac{\partial^2 f_3}{\partial x_1 \partial x_3} \right)_{E_0} = \beta\sigma^*, \\ \left(\frac{\partial^2 f_3}{\partial x_2 \partial x_3} \right)_{E_0} &= \gamma\sigma^*, \quad \left(\frac{\partial^2 f_3}{\partial x_3^2} \right)_{E_0} = -2a - 2\beta\sigma^* \left(\frac{\kappa}{\vartheta + \xi} \right) - 2\gamma\sigma^* \left(\frac{\kappa\xi}{\vartheta(\vartheta + \xi)} \right), \\ \text{and } \left(\frac{\partial^2 f_3}{\partial x_3 \partial \sigma^*} \right)_{E_0} &= \frac{\beta\kappa\vartheta + \gamma\kappa\xi}{\vartheta(\vartheta + \xi)}.\end{aligned}$$

Thus, the coefficients a_1 and b_1 are computed as

$$a_1 = -\frac{2(a\vartheta^2(\vartheta + \xi)^2 + \kappa\sigma^*(\beta^2\vartheta^2\sigma^* + \gamma\xi(\vartheta + \xi)(\vartheta + \gamma\sigma^*) + \beta\vartheta(\vartheta^2 + \vartheta\xi + \gamma\xi\sigma^*)))}{\vartheta^2(\vartheta + \xi)^2}, \quad (17)$$

$$b_1 = \frac{\beta\kappa\vartheta + \gamma\kappa\xi}{\vartheta(\vartheta + \xi)}. \quad (18)$$

By Theorem 4.1 given by Castillo-Chavez and Song [7], the local dynamics of system (16) around E_0 at $R_0 = 1$ is completely determined by the signs of bifurcation

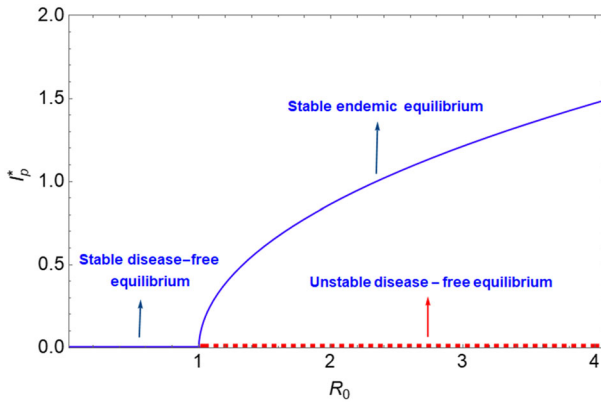


Fig. 2 R_0 vs. I_p^*

coefficients a_1 , and b_1 . More precisely, if $a_1 < 0$ and $b_1 > 0$, then the bifurcation is forward; if $a_1 > 0$ and $b_1 > 0$ then the bifurcation is backward. Since, the bifurcation coefficients obtained in Eqs. (17-18) shows that a_1 is always negative, and b_1 is always positive, therefore, the bifurcation is forward. “In the forward bifurcation [22], when $R_0 < 1$, a small influx of infected individuals will not generate large outbreaks, and the disease dies out in time, and the corresponding disease-free equilibrium is asymptotically-stable. On the other hand, when R_0 exceeds unity, then the disease will persist with a stable endemic equilibrium. This phenomenon, where the disease-free equilibrium loses its stability and a stable endemic equilibrium appears as the basic reproduction number increases through one, is known as forward bifurcation” The main characteristics of forward bifurcation are :

1. There is no endemic equilibrium near disease-free equilibrium when $R_0 < 1$. That is, the disease-free equilibrium is often the only equilibrium when $R_0 < 1$.
2. A low level of endemicity when R_0 is slightly above unity.

Thus, the following theorem is concluded:

Theorem 3 *The disease-free equilibrium E_0 at $R_0 = 1$ of the system (6) exhibits a forward bifurcation at E_0 and $R_0 = 1$ when $\varpi = 0$.*

Figure 2 illustrates the result of Theorem 3 for the following numerical experimental data: $\kappa = 2$, $\beta = 0.09$, $\xi = 0.12$, $\sigma = 0.08$, $\vartheta = 0.01$, $\gamma = 0.009$, $d = 0.08$, $\theta = 0.03$, $a = 0.005$, $b = 0.01$, $c = 0.03$. At these values of parameters, bifurcation constants $a_1 = -0.543519 < 0$ and $b_1 = 3.04615 > 0$, which satisfies the theoretical condition for the occurrence of forward bifurcation. This figure provides the qualitative description of infectives when R_0 varies from unity. It depicts that when R_0 crosses unity from below, a small positive asymptotically stable endemic equilibrium exists, and E_0 changes its stability from stable to unstable.

4.2 Existence and local stability of the endemic equilibrium

Now, we determine the conditions for endemic equilibrium existence. For that, assuming that $X_p, A_p, I_p \neq 0$ and setting the system (6) to zero, we get:

$$\kappa - \vartheta X_p - \frac{\beta\sigma X_p I_p}{1 + I_p} - \xi X_p = 0, \quad (19)$$

$$\xi X_p - \vartheta A_p - \frac{\gamma\sigma A_p I_p}{1 + I_p} = 0, \quad (20)$$

$$\frac{\beta\sigma X_p I_p}{1 + I_p} + \frac{\gamma\sigma A_p I_p}{1 + I_p} - (\vartheta + d + \theta)I_p - \frac{aI_p^2}{bI_p^2 + cI_p + 1} = 0. \quad (21)$$

On obtaining X_p from Eq. (19) and A_p from Eq. (20), and then substituting it in (21), the following quartic equation in I_p is obtained:

$$F(I_p) := K_0 + K_1 I_p + K_2 I_p^2 + K_3 I_p^3 + K_4 I_p^4 = 0, \quad (22)$$

where,

$$\begin{aligned} K_0 &= \vartheta(\vartheta + \xi)(d + \vartheta + \theta)(1 - R_0), \\ K_1 &= a\vartheta(\xi + \vartheta) + (d + \theta + \vartheta)(\sigma\vartheta(\beta + \gamma) + \gamma\xi\sigma + (\xi + \vartheta)(c(1 - R_0)\vartheta + \vartheta)) \\ &\quad + (\vartheta(\xi + \vartheta)(d + \theta + \vartheta) - \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)), \\ K_2 &= a\sigma(\beta\vartheta + \gamma(\xi + \vartheta)) + 2a\vartheta(\xi + \vartheta) + b(1 - R_0)\vartheta(\xi + \vartheta)(d + \theta + \vartheta) \\ &\quad + (d + \theta + \vartheta)(\beta\gamma\sigma^2 + (c + 1)\sigma(\beta\vartheta + \gamma(\xi + \vartheta)) + (c + 1)\vartheta(\xi + \vartheta)) \\ &\quad + c(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) - \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)), \\ K_3 &= a(\gamma\sigma + \vartheta)(\beta\sigma + \xi + \vartheta) + (d + \theta + \vartheta)(\sigma(b + c)(\beta\vartheta + \gamma(\xi + \vartheta)) \\ &\quad + \vartheta(b + c)(\xi + \vartheta) + \beta c \gamma \sigma^2) \\ &\quad + b(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) - \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)), \\ K_4 &= b(\sigma\gamma + \vartheta)(d + \theta + \vartheta)(\sigma\beta + \vartheta + \xi). \end{aligned} \quad (23)$$

For the positive root I_p^* of polynomial $F(I_p)$, we can make

$$X_p^* = \frac{(1 + I_p^*)\kappa}{\sigma I_p^* \beta + (1 + I_p^*)(\vartheta + \xi)} > 0. \quad (24)$$

and

$$A_p^* = \frac{(1 + I_p^*)^2 \kappa \xi}{(\sigma I_p^* \gamma + \vartheta + I_p^* \vartheta)(\sigma I_p^* \beta + (1 + I_p^*)(\vartheta + \xi))} > 0. \quad (25)$$

So, $E_e(X_p^*, A_p^*, I_p^*)$ is an endemic equilibrium of (6).

In this paper, we consider the case of the existence of a unique endemic equilibrium only.

Theorem 4 *When $R_0 < 1$, the system (6) has no endemic equilibrium if*

$$(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) > \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)).$$

Proof Let $R_0 < 1$, which implies $1 - R_0 > 0$, and so $K_0 > 0$. Also, $K_4 > 0$. The coefficients K_2 , K_3 , and K_4 are positive if

$$(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) - \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)) > 0$$

Or

$$(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) > \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi)).$$

Hence, by Descartes' rule of signs [44], Eq. (22) has no positive root under the condition $(\vartheta(\xi + \vartheta)(d + \theta + \vartheta) > \kappa\sigma(\beta(\gamma\sigma + \vartheta) + \gamma\xi))$ and so the system (6) has no positive equilibrium under this condition. \square

Theorem 5 *When $R_0 > 1$, there exists either a unique or three endemic equilibria if all equilibria are simple roots.*

Proof Let $R_0 > 1$. Then, the coefficient $K_0 < 0$. Also, $K_4 > 0$. From Eq. (22), we have

$$F(I_p) := K_0 + K_1 I_p + K_2 I_p^2 + K_3 I_p^3 + K_4 I_p^4 = 0.$$

The conditions U_1 – U_8 , given below, define the possibilities for the signs of K_1 , K_2 , and K_3 as follows:

$$\begin{aligned} U_1 &: K_1 > 0, K_2 > 0, \text{ and } K_3 > 0, \\ U_2 &: K_1 < 0, K_2 < 0, \text{ and } K_3 > 0, \\ U_3 &: K_1 < 0, K_2 > 0, \text{ and } K_3 > 0, \\ U_4 &: K_1 < 0, K_2 < 0, \text{ and } K_3 < 0, \\ U_5 &: K_1 > 0, K_2 > 0, \text{ and } K_3 < 0, \\ U_6 &: K_1 > 0, K_2 < 0, \text{ and } K_3 > 0, \\ U_7 &: K_1 > 0, K_2 < 0, \text{ and } K_3 < 0, \\ U_8 &: K_1 < 0, K_2 > 0, \text{ and } K_3 < 0. \end{aligned} \tag{26}$$

By Descartes' rule of signs [44], under the conditions U_1 – U_8 , the equation $F(I_p)$ can have either a unique or three positive roots. Under any of the conditions U_1 – U_4 , there exists a unique endemic equilibrium, and under U_5 – U_8 , there exists either a unique or three endemic equilibria.

Thus, we state the following theorem: \square

Theorem 6 *Assume that any of the conditions $H1$: (U_1 – U_8), and $R_0 > 1$ hold, then the system (6) has a unique endemic equilibrium.*

Next, we examine the stability behavior of the unique endemic equilibrium. At E_e , the characteristic equation of the system (6) is obtained as

$$\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0 + (M_2\lambda^2 + M_1\lambda + M_0)e^{-\lambda\varpi} + (N_1\lambda + N_0)e^{-2\lambda\varpi} = 0, \quad (27)$$

where,

$$L_2 = d + \frac{aI_p^*(2 + cI_p^*)}{(1 + I_p^*(c + bI_p^*))^2} + \theta + 3\vartheta + \xi,$$

$$L_1 = 2\theta\vartheta + 3\vartheta^2 + \theta\xi + 2\vartheta\xi + d(2\vartheta + \xi) + \frac{a(2\vartheta + \xi)I_p^*(2 + cI_p^*)}{(1 + I_p^*(c + bI_p^*))^2},$$

$$L_0 = \vartheta \left(d + \frac{aI_p^*(2 + cI_p^*)}{(1 + I_p^*(c + bI_p^*))^2} + \theta + \vartheta \right) (\vartheta + \xi),$$

$$M_2 = \frac{(-X_p^*\beta - A_p^*\gamma + I_p^*(1 + I_p^*)(\beta + \gamma))\sigma}{(1 + I_p^*)^2},$$

$$M_1 = \frac{aI_p^{*2}(2 + cI_p^*)(\beta + \gamma)\sigma}{(1 + I_p^*)(1 + I_p^*(c + bI_p^*))^2} + \frac{I_p^*((\beta + \gamma)(d + \theta + 2\vartheta) + \gamma\xi)\sigma}{1 + I_p^*}$$

$$- \frac{(X_p^*\beta + A_p^*\gamma)(2\vartheta + \xi)\sigma}{(1 + I_p^*)^2},$$

$$M_0 = \frac{I_p^*(d + \theta + \vartheta)(\beta\vartheta + \gamma(\vartheta + \xi))\sigma}{1 + I_p^*} + \frac{aI_p^{*2}(2 + cI_p^*)(\beta\vartheta + \gamma(\vartheta + \xi))\sigma}{(1 + I_p^*)(1 + I_p^*(c + bI_p^*))^2}$$

$$- \frac{(X_p^*\beta + A_p^*\gamma)\vartheta(\vartheta + \xi)\sigma}{(1 + I_p^*)^2},$$

$$N_1 = \frac{I_p^*(-A_p^* - X_p^* + I_p^* + I_p^{*2})\beta\gamma\sigma^2}{(1 + I_p^*)^3},$$

$$N_0 = \frac{I_p^*\beta\gamma(-(A_p^* + X_p^*)\vartheta + I_p^*(1 + I_p^*)(d + \theta + \vartheta))\sigma^2}{(1 + I_p^*)^3}$$

$$+ \frac{aI_p^{*3}(2 + cI_p^*)\beta\gamma\sigma^2}{(1 + I_p^*)^2(1 + I_p^*(c + bI_p^*))^2}.$$

Multiplying Eq. (27) by $e^{\lambda\varpi}$, we get

$$M_2\lambda^2 + M_1\lambda + M_0 + (\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0)e^{\lambda\varpi} + (N_1\lambda + N_0)e^{-\lambda\varpi} = 0. \quad (28)$$

When $\varpi = 0$, Eq. (28) becomes

$$\lambda^3 + L_{00}\lambda^2 + L_{01}\lambda + L_{02} = 0, \quad (29)$$

where,

$$\begin{aligned} L_{00} &= M_2 + L_2, \\ L_{01} &= M_1 + L_1 + N_1, \\ L_{02} &= M_0 + L_0 + N_0. \end{aligned}$$

By Routh-Hurwitz criterion, Eq. (29) has all the roots with negative real parts under **H2**., given below:

$$\mathbf{H2:} \quad L_{00} > 0, \quad L_{02} > 0, \quad \text{and} \quad L_{00}L_{01} > L_{02}. \tag{30}$$

Hence, we state the following Theorem:

Theorem 7 *Assume that **H2** holds. Then, the endemic equilibrium E_e is locally asymptotically stable when $\varpi = 0$.*

Now, for $\varpi > 0$, let Eq. (28) has a root $i\omega$ ($\omega > 0$). Then, replacing λ with $i\omega$ ($\omega > 0$) in Eq. (28) and splitting real and imaginary parts, we obtain

$$B_1(\omega) \cos \omega\varpi - B_2(\omega) \sin \omega\varpi = B_3(\omega), \tag{31}$$

$$B_4(\omega) \sin \omega\varpi + B_5(\omega) \cos \omega\varpi = B_6(\omega). \tag{32}$$

where,

$$\begin{aligned} B_1(\omega) &= -L_2\omega^2 + L_0 + N_0, \\ B_2(\omega) &= \omega(L_1 - N_1) - \omega^3, \\ B_3(\omega) &= M_2\omega^2 - M_0, \\ B_4(\omega) &= -L_2\omega^2 + L_0 - N_0, \\ B_5(\omega) &= (L_1 + N_1)\omega - \omega^3, \\ B_6(\omega) &= -M_1\omega. \end{aligned} \tag{33}$$

From Eqs. (31) and (32), we obtain

$$\sin \omega\varpi = \frac{B_{01}(\omega)}{B_{00}(\omega)}, \tag{34}$$

$$\cos \omega\varpi = \frac{B_{02}(\omega)}{B_{00}(\omega)}, \tag{35}$$

where,

$$\begin{aligned} B_{00} &= L_0^2 - N_0^2 + (L_1^2 - 2L_0L_2 - N_1^2)\omega^2 + (-2L_1 + L_2^2)\omega^4 + \omega^6, \\ B_{01} &= (L_1M_0 - L_0M_1 - M_1N_0 + M_0N_1)\omega + (-M_0 + L_2M_1 - L_1M_2 - M_2N_1)\omega^3 + M_2\omega^5, \\ B_{02} &= -L_0M_0 + M_0N_0 + (L_2M_0 - L_1M_1 + L_0M_2 - M_2N_0 + M_1N_1)\omega^2 + (M_1 - L_2M_2)\omega^4. \end{aligned} \tag{36}$$

On squaring and then adding Eqs. (34) and (35), we obtain

$$B_{01}^2(\omega) + B_{02}^2(\omega) - B_{00}^2(\omega) = 0. \tag{37}$$

Let **(H3):** There exists at least one positive root ω_0 of Eq. (37), i.e., Eq. (28) has a pair of purely imaginary roots $\pm i\omega_0$.

For ω_0 , the corresponding critical value of the time delay ϖ_k is obtained as follows:

$$\varpi_k = \frac{1}{\omega_0} \arccos\left(\frac{B_{02}(\omega_0)}{B_{00}(\omega_0)}\right) + \frac{2k\pi}{\omega_0}, \quad k = 0, 1, 2, 3, \dots \tag{38}$$

Let $\varpi_0 = \min\{\varpi_k, k = 0, 1, 2, 3, \dots\}$.

To establish Hopf bifurcation, we must have $Re \left[\frac{d\lambda}{d\varpi} \right]^{-1} \Big|_{\lambda=i\omega_0} \neq 0$.

Differentiating Eq. (28) with respect to ϖ yields

$$\left[\frac{d\lambda}{d\varpi} \right]^{-1} = \frac{2\lambda M_2 + M_1 + N_1 e^{-\lambda\varpi} + e^{\lambda\varpi} (3\lambda^2 + 2\lambda L_2 + L_1)}{\lambda ((N_1\lambda + N_0)e^{-\lambda\varpi} - (\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0)) e^{\lambda\varpi}} - \frac{\varpi}{\lambda}.$$

From Eq. (28), we have

$$-(\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0)e^{\lambda\varpi} = M_2\lambda^2 + M_1\lambda + M_0 + (N_1\lambda + N_0)e^{-\lambda\varpi}.$$

Thus, we obtain

$$\left[\frac{d\lambda}{d\varpi} \right]^{-1} = \frac{2\lambda M_2 + M_1 + N_1 e^{-\lambda\varpi} + e^{\lambda\varpi} (3\lambda^2 + 2\lambda L_2 + L_1)}{\lambda (2(N_1\lambda + N_0)e^{-\lambda\varpi} + (M_2\lambda^2 + M_1\lambda + M_0))} - \frac{\varpi}{\lambda}.$$

So,

$$Re \left[\frac{d\lambda}{d\varpi} \right]^{-1} \Big|_{\lambda=i\omega_0} = \frac{Y}{P^2 + Q^2},$$

where,

$$\begin{aligned} Y &= \omega_0(-M_1^2\omega_0 + 2M_0M_2\omega_0 - 2N_1^2\omega_0 - 2M_2^2\omega_0^3 + \omega_0(-L_1M_1 + 4M_2N_0 \\ &\quad - 3M_1N_1 + 3M_1\omega_0^2 \\ &\quad + 2L_2(M_0 - M_2\omega_0^2)) \cos \varpi\omega + 2\omega(2L_2N_0 - L_1N_1 \\ &\quad + 3N_1\omega_0^2) \cos 2\varpi\omega + (-M_0N_1 - 3M_0\omega_0^2 \\ &\quad + 5M_2N_1\omega_0^2 + 3M_2\omega_0^4 + 2M_1(N_0 + L_2\omega_0^2) + L_1(M_0 - M_2\omega_0^2)) \sin \varpi\omega + 2(L_1N_0 \\ &\quad + (-3N_0 + 2L_2N_1)\omega_0^2) \sin 2\varpi\omega), \\ P &= -M_1\omega_0^2 - 2N_1\omega_0^2 \cos \varpi\omega_0 + 2N_0\omega_0 \sin \varpi\omega_0, \\ Q &= M_0\omega_0 - M_2\omega_0^3 + 2N_0\omega_0 \cos \varpi\omega_0 + 2N_1\omega_0^2 \sin \varpi\omega_0. \end{aligned}$$

Obviously, if **H4**: $Y \neq 0$, then $Re \left[\frac{d\lambda}{d\varpi} \right]^{-1} \Big|_{\lambda=i\omega_0} \neq 0$.

Thus, the following theorem is stated:

Theorem 8 Suppose **(H1-H4)** holds. Then, the endemic equilibrium $E_e(X_p^*, A_p^*, I_p^*)$ of the system (6) is

1. locally asymptotically stable when $\varpi \in [0, \varpi_0)$,
2. undergoes a Hopf bifurcation when $\varpi = \varpi_0$, and a family of periodic solutions bifurcate from $E_e(X_p^*, A_p^*, I_p^*)$ when ϖ crosses ϖ_0 .

4.3 Global stability

In this subsection, we establish the global stability behavior of the DFE and EE.

4.3.1 Global stability behavior of disease-free equilibrium

We examine the global stability behavior of DFE $E_0(X_0, A_0, I_0) = E_0\left(\frac{\kappa}{\vartheta+\xi}, \frac{\kappa\xi}{\vartheta(\vartheta+\xi)}, 0\right)$ for $R_0 \leq 1$ by constructing a suitable Lyapunov function. For this, the following postulates are proposed:

P4. $\Psi'_{I_p}(X_p(t), 0)$ is increasing for $X_p(t) > 0$ and $\Lambda'_{I_p}(A_p(t), 0)$ is increasing for $A_p(t) > 0$.

P5. $\frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} < 1$ for $X_p(t) > X_0$, $\frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} > 1$ for $X_p(t) \in (0, X_0)$,
 $\frac{\Lambda'_{I_p}(A_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} < 1$ for $A_p(t) > A_0$, $\frac{\Lambda'_{I_p}(X_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} > 1$ for $A_p(t) \in (0, A_0)$.

P6.

$$\begin{aligned} & \Phi(X_p(t), I_p(t)) + \Lambda(A_p(t), I_p(t)) \\ & \leq I_p(t) \left(\left(\frac{\partial \Phi(X_p(t), I_p(t))}{\partial I_p} \right)_{(X_0, 0)} + \left(\frac{\partial \Lambda(A_p(t), I_p(t))}{\partial I_p} \right)_{(A_0, 0)} \right) - \left(\frac{\partial H(I_p)}{\partial I_p} \right)_{I_p=0} \\ & \quad + H(I_p(t)), \text{ where } H(I_p(t)) = (\vartheta + d + \zeta)I_p(t) + h(I_p(t)) \text{ and } I_p(t) > 0. \end{aligned}$$

P7. $\frac{X_p}{X_0} > \frac{A_p}{A_0}$ if $\frac{X_p}{X_0} > 1$ and $\frac{X_p}{X_0} < \frac{A_p}{A_0}$ if $\frac{X_p}{X_0} < 1$.

Under these postulates, the following theorem is being proposed:

Theorem 9 Suppose that (P1.)–(P7.) and $R_0 \leq 1$ hold. Then, DFE $E_0(X_0, A_0, 0)$ of the system (6) is globally asymptotically stable for $\varpi \geq 0$.

The proof has been given in Appendix.

4.3.2 Global stability behavior of endemic equilibrium

Now, we examine the global stability behavior of $E_e(X^*, A^*, I^*)$ of the system (6) by constructing a Lyapunov functional and employing the Lyapunov direct method. To proceed, we propose the following postulates:

P8.

$$\begin{aligned} & \left(\frac{\Phi(X_p^*, I_p^*)}{\Phi(X_p(t), I_p^*)} - \frac{I_p^*}{I_p(t)} \right) \leq 0; \quad \left(\frac{\Phi(X_p(t), I_p(t))}{\Phi(X_p^*, I_p^*)} - 1 \right) \leq 0; \\ & \left(\frac{\Phi(X_p(t), I_p^*)}{\Phi(X_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*} \right) \leq 0; \\ & \left(\frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} - \frac{I_p^*}{I_p(t)} \right) \leq 0; \quad \left(\frac{\Lambda(A_p(t), I_p(t))}{\Lambda(A_p^*, I_p^*)} - 1 \right) \leq 0; \\ & \left(\frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*} \right) \leq 0 \text{ for } I_p \geq I_p^*. \end{aligned}$$

P9.

$$\left(\frac{h(I_p^*)}{h(I_p(t))} - \frac{I_p^*}{I_p(t)} \right) \left(\frac{I_p(t)}{I_p^*} - 1 \right) \leq 0 \text{ for } I_p \geq I_p^*.$$

P10.

$$\frac{X_p}{X_p^*} - \frac{A_p}{A_p^*} > 0 \text{ for } \frac{X_p}{X_p^*} > 1 \text{ and } \frac{X_p}{X_p^*} - \frac{A_p}{A_p^*} < 0 \text{ for } \frac{X_p}{X_p^*} < 1.$$

The following theorem is proposed under these postulates:

Theorem 10 Suppose that (P1.)–(P3.), (P8.)–(P10.), and $R_0 > 1$ hold. Then, the EE $E_e(X_p^*, A_p^*, I_p^*)$ of the system (6) is globally asymptotically stable for $\varpi \geq 0$.

The proof has been given in Appendix.

5 Numerical simulation

This section aims to validate the theoretical findings which we have discussed in previous sections and show the significance of various parameters by considering numerical experimental data. For this, we assume that all the parameters are positive. Two examples have been considered herein:-

1. Example 1 illustrates the existence of a unique endemic equilibrium and its stability. Also, it shows the significance of considering various parameters such as time delay, treatment rate, awareness rate, and the incorporation of the aware compartment in the SIR epidemic model.
2. Example 2 illustrates the occurrence of Hopf bifurcation via oscillatory and periodic solutions.

Example 1 We consider the following experimental data: $\kappa = 2$, $\beta = 0.09$, $\xi = 0.12$, $\sigma = 1$, $\vartheta = 0.01$, $\gamma = 0.009$, $d = 0.08$, $\theta = 0.03$, $a = 0.005$, $b =$

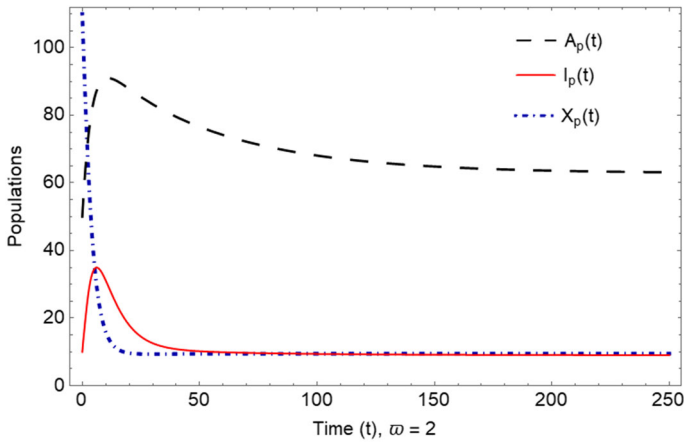


Fig. 3 The temporal behavior of susceptible, alert, and infected populations for time-delay $\varpi = 2$

0.01, $c = 0.03$. At these values of parameters, we calculate that $R_0 = 25.3846 > 1$ and the coefficients of the polynomial $F(I_p) := K_0 + K_1 I_p + K_2 I_p^2 + K_3 I_p^3 + K_4 I_p^4 = 0$, given in Eq. (22) have the value $K_0 = -0.003804 < 0$, $K_1 = -0.00512722 < 0$, $K_2 = 0.000336322 > 0$, $K_3 = -0.000014248 < 0$, and $K_4 = 5.016 * 10^{-6} > 0$. So, one of the conditions U_8 given in Eq. (26) is satisfied. Further, the solutions of the polynomial equation $F(I_p) = 0$ are $I_p = -2.73341 - 10.5534i$, $-2.73341 + 10.5534i$, -0.707827 and 9.01515 . So, the only positive root of $F(I_p) = 0$ is $I_p^* = 9.01515$. Thus, the conditions of Theorem 6 are satisfied and there exists a unique endemic equilibrium $E_e(9.47806, 62.8333, 9.01515)$.

Figure 3 shows the qualitative behavior of susceptible, alert, and infected populations for a time delay $\varpi = 2$. It is depicted that the susceptible population decreases over time and a large population gets aware of the disease, and as time passes, they become less serious, and finally both the populations settle down to a steady state. Also, it elucidates that infectives increase at a high rate and then start decreasing and eventually reach their steady state.

Figure 4 indicates the impact of latent period ϖ on infected individuals $I_p(t)$. We can see the variation in the number of infectives for higher values of time delay. This figure confirms that the longer the latent period (time delay) of infection, the higher its spread, which shows the importance of considering time delay in studying infectious disease’s dynamical behavior.

Figure 5 shows the influence of transmission rates β of susceptibles and γ of aware individuals on infectives. The higher the transmission rates, the higher the possibility of spreading infection. Therefore, it is imperative to minimize the interaction rate of susceptible and aware people with infected individuals.

Figure 6 shows the power of cure rate (a) in declining the number of infected individuals. An increased cure rate can reduce the infection level efficiently. Thus, the accessibility of treatment resources and adequate treatment is significant in declining the cases of infected individuals.

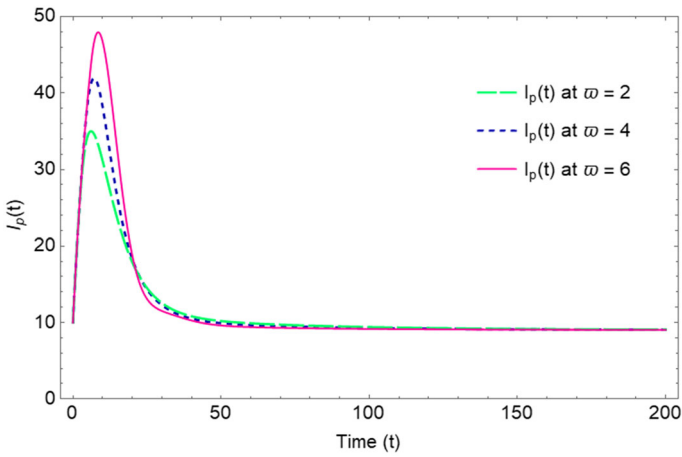


Fig. 4 Infected population $I_p(t)$ when time delay ϖ varies

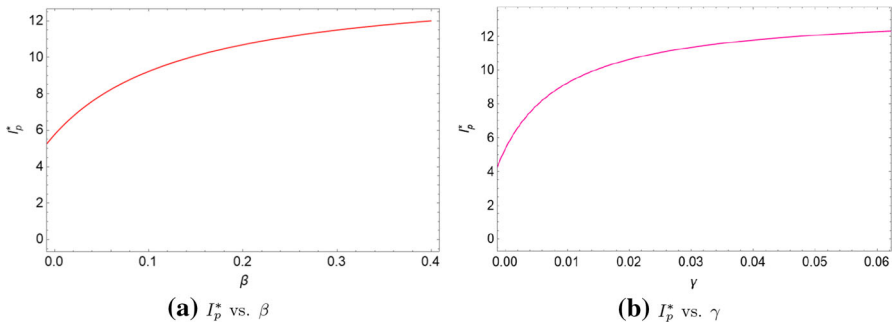


Fig. 5 Variation in the number of infected individuals when transmission rates β and γ vary

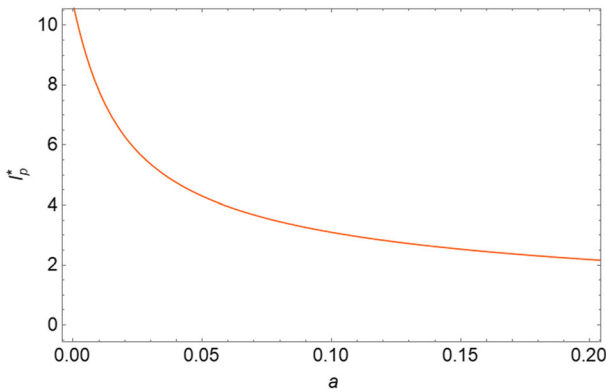


Fig. 6 Effect of cure rate (a) on I_p^*

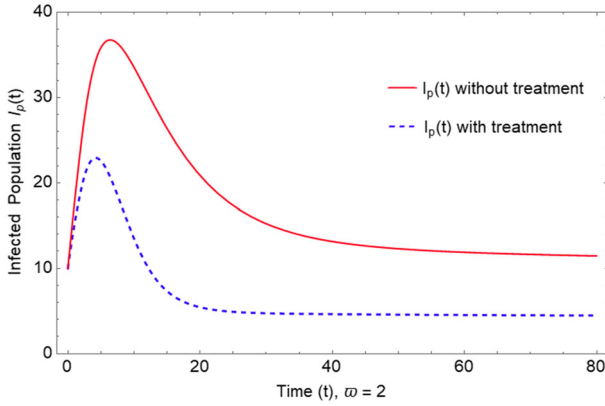


Fig. 7 Effect of saturating treatment rate ($H(I_p(t))$) on infected population ($I_p(t)$)

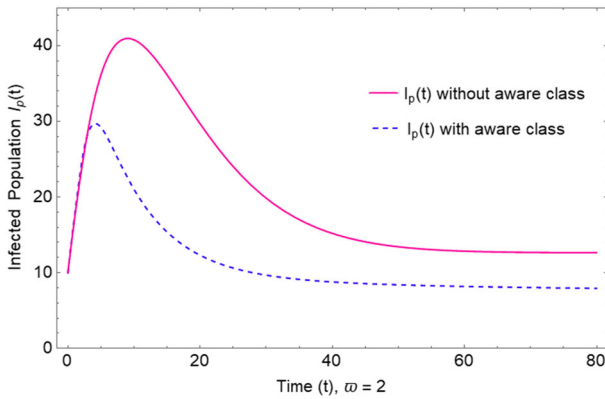


Fig. 8 Effect of incorporating awareness class on the infected population $I_p(t)$ for $\omega = 2$

Figure 7 demonstrates the impact of nonlinear saturating treatment rate on infected individuals. The infected population, drawn for the $a = 0.05$, reveals the significance of the availability of treatment to infected people. If the health system has sufficient treatment facilities, then the spread of infection can be controlled on a large scale.

Figure 8 shows the difference in the number of infected individuals with the inclusion and exclusion of aware individuals' compartment, deliberating that unaware individuals are becoming infected faster than those individuals who are familiar with the disease spread. It shows the relationship between human awareness and the spread of infection. The graph of infected individuals with awareness class is drawn for the awareness rate $\xi = 0.6$, revealing that a considerable value of awareness rate causes more individuals to be safe from illnesses. The disease spread awareness alerts people and helps them to take necessary protection measures against disease which reduce the occurrence of infection on a large scale. Thus, the significance of incorporating an alert compartment in the SIR model is vital.

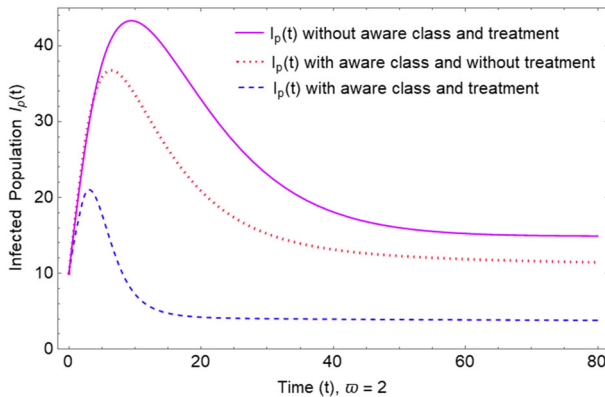


Fig. 9 Effect of both awareness class $A_p(t)$ and saturated treatment rate on $I_p(t)$ for $\varpi = 2$

Figure 9 depicts the number of the infected population for the cases: neither awareness nor treatment is available (shown by the solid purple line); people are aware of the disease, but treatment for infectives is not available (indicated by the dotted red line); and when both treatment and awareness are present (indicated through the dashed blue line). This figure captures the impact of both awareness, and treatment on infectives. When awareness and treatment are absent, the infected population stabilizes at a high level. If the treatment is not available, then the awareness among people reduces the spread of infection with a big difference. For eradicating disease or lowering it to the lowest level, the presence of both awareness among susceptibles and sufficient availability of treatment resources has a vital role.

Example 2 The following numerical experimental data is considered to validate the occurrence of Hopf bifurcation:

$$\begin{aligned} \kappa = 5, \quad \xi = 0.001, \quad \vartheta = 0.01, \quad \beta = 0.009, \quad \gamma = 0.001, \quad \sigma = 10. \\ d = 0.08, \quad \theta = 0.03, \quad a = 0.005, \quad b = 0.01, \quad c = 0.03. \end{aligned}$$

At these values of parameters, it has been verified that Eq. (37) has a unique positive root $\omega_0 = 0.08524468136097717$. For this value of ω_0 , the corresponding critical value of the time delay ϖ_0 given in the formula of Eq. (38), is obtained as $\varpi_0 = 19.7113$.

Figure 10 shows the time series solution and respective phase portraits of susceptible, aware and infected populations. Figures. 10(a)-10(d) have been plotted for $\varpi = 17$, and $\varpi = 19$, which reveals that the endemic equilibrium is stable when the time delay is less than its critical value $\varpi_0 = 19.7113$. On the other hand, Figs. 10(e) and 10(f) have been plotted for the time delay $\varpi = 19.8$ which reveals that the orbits spiral goes away from endemic equilibrium as ϖ crosses its critical value $\varpi_0 = 19.7113$, and the endemic equilibrium turns into unstable. These figures confirm the result of the Theorem 8.

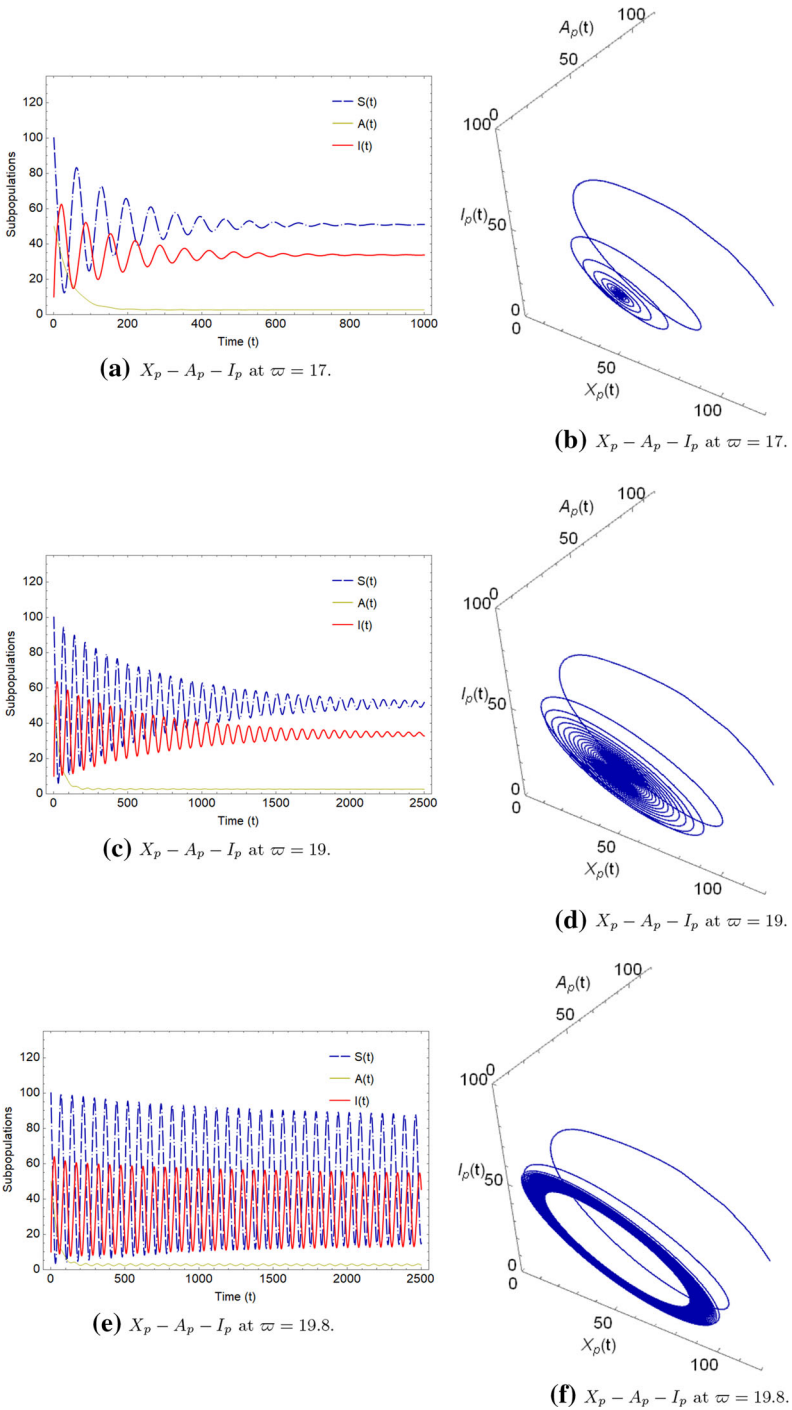


Fig. 10 The time series solutions and respective phase portraits of susceptible, aware and infected subpopulations

6 Discussion

During an outbreak, awareness about the transmission routes and interventions of a disease can alert individuals regarding the infection risk, resulting in a change in human behavior and disease transmission patterns. Therefore, the present article studies a mathematical epidemic model with awareness effects to study disease transmission and control dynamics. We comprised four dynamical variables in our model: susceptible, aware, infected, and recovered individual; and proposed a nonlinear time-delayed SAIR epidemic model by incorporating Michaelis-Menten type incidences with latent period and a saturating treatment rate. We assume that aware individuals can also catch the infection, probably at a lower rate than the fully vulnerable population. We analyze the model mathematically, revealing that it has two equilibria: the disease-free equilibrium (complete eradication of infection) and the endemic equilibrium (persistence of disease at a certain level). We obtain the model's threshold quantity, the basic reproduction number R_0 , and perform the stability analysis to determine whether the disease eliminates or persists. The basic reproduction number determines the potential for an infectious agent to start an outbreak, the degree of transmission without control measures, and the capacity of control measures to diminish spread. The delayed system analysis reveals that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, unstable when $R_0 > 1$, and neutrally linearly stable when $R_0 = 1$. However, using the center manifold theory approach, we show that the undelayed system exhibits a forward bifurcation at $R_0 = 1$, meaning that reducing R_0 below unity is enough to eradicate society's infection. Further, we investigate the endemic equilibrium's local stability, and prove the existence of oscillating and periodic solutions near-endemic equilibrium through Hopf bifurcation, concerning time delay as the bifurcation parameter. Furthermore, the global stability behavior of the disease-free and endemic equilibria is also examined using the Lyapunov functionals by employing the Lyapunov method. It is shown that the disease-free equilibrium is globally asymptotically stable when $R_0 < 1$, and the endemic equilibrium is globally asymptotically stable when $R_0 > 1$.

The numerical simulations validate the effectiveness of theoretical findings and show the impact of the model's parameters. It is observed that the longer the time delay, the higher the number of individuals who catch the infection. The oscillatory solutions for various values of time delay establish Hopf bifurcations near-endemic equilibrium. Moreover, if the time delay crosses its critical value, then the trajectories of the solutions bifurcate from endemic equilibrium and destabilize the system. We show that the number of infected individuals is much higher in the SIR model (i.e., without awareness) in comparison to the number of infected in the SAIR model (i.e., with awareness). If susceptible individuals are aware of infection risk, they will be on high alert and choose not to go to crowded areas, avoid unnecessary contact with infected individuals, and implement other anti-epidemic inhibition measures which reduce the infection spread effectively. The numerical result shows the impact of saturating treatment rate, which reveals that adequate treatment availability is crucial in controlling infection spread. If the treatment facilities are not enough, individuals' awareness and their willingness to adopt anti-epidemic measures are the only way to

reduce infection. Individuals' awareness together with sufficient treatment facilities for infectives can reduce or even eradicate the infectious disease from society.

The present study consisting of nonlinear incidences of unaware and aware susceptibles with the latent period, and saturated treatment rate, signifies the substantial role of the latent period in the disease transmission process, susceptibles' behavior in preventing disease spread, and limitation in treatment facilities in curing infectives. The results indicate that awareness about the spread of infection in susceptible individuals is vital in preventing disease transmission and is a potential policy for controlling the disease spread in the absence of treatment availabilities. The public health authorities and the government have a significant contribution to raising awareness among people and encouraging them to adopt anti-epidemic measures. For example, the government is enforcing different non-pharmaceutical interventions to obstruct COVID-19 transmission due to the absence of proper therapeutics or vaccines. Several countries focus on raising awareness through media advertising campaigns to encourage people to maintain social distance, wear a face mask, adopt healthy sanitation practices, wear hand gloves, avoid touching surfaces, regular hand washing, etc. These behaviors urge people to adopt preventive measures, reduce contact with others, and so reduce disease spread, consequently suppressing disease spread burden. Thus, awareness about the disease with the encouragement of adopting preventive measures and appropriate treatment facilities for infectives can efficiently reduce disease spread.

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Declarations

Conflicts of interest The authors declare that they have no conflict of interest.

Appendix

Proof of Theorem 9

Proof (P1.) and (P2.) establish that $E_0(X_0, A_0, 0)$ is the only equilibrium of the system (6).

The Lyapunov functional is given as

$$V(t) = V_1(t) + V_2(t),$$

where,

$$\begin{aligned} V_1(t) &= X_p(t) + A_p(t) - X_0 - A_0 - \int_{X_0}^{X_p(t)} \lim_{I_p \rightarrow 0^+} \frac{\Psi(X_0, I_p(t))}{\Psi(\varepsilon, I_p(t))} d\varepsilon \\ &\quad - \int_{A_0}^{A_p(t)} \lim_{I_p \rightarrow 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(\varepsilon, I_p(t))} d\varepsilon + I_p(t), \\ V_2(t) &= \int_0^w (\Psi(X_p(t - \rho), I_p(t - \rho)) + \Lambda(A_p(t - \rho), I_p(t - \rho))) d\rho. \end{aligned}$$

Using the postulates (P1.)–(P3.), it follows that $V_1(t)$ is well-defined and continuously differentiable function $\forall X_p(t) > 0$, $A_p(t) > 0$, $I_p(t) > 0$, and $V(t) = 0$ at $E_0(X_0, A_0, 0)$. Now, we show that $\frac{dV(t)}{dt} \leq 0$ for all $t \geq 0$. First, we compute $\frac{dV_1(t)}{dt}$ as follows:

$$\begin{aligned} \frac{dV_1(t)}{dt} &= \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Psi(X_0, I(t))}{\Psi(X_p, I_p(t))}\right) X'_p(t) + \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) A'_p(t) + I'_p(t) \\ &= \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Psi(X_0, I_p(t))}{\Psi(X_p, I_p(t))}\right) (\kappa - \vartheta X_p - \Psi(X_p(t - \varpi), I_p(t - \varpi)) - \xi X_p) \\ &\quad + \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) (\xi X_p - \vartheta A_p - \Lambda(A_p(t - \varpi) I_p(t - \varpi))) \\ &\quad + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\ &\quad + \Lambda(A_p(t - \varpi) I_p(t - \varpi)) - (\vartheta + d + \theta) I_p - \frac{a I_p^2}{b I_p^2 + c I_p + 1}. \end{aligned}$$

Since $\kappa - (\vartheta + \xi) X_p = (\vartheta + \xi)(X_0 - X_p)$, thus, we obtain:

$$\begin{aligned} \frac{dV_1(t)}{dt} &= \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Psi(X_0, I_p(t))}{\Psi(X_p, I_p(t))}\right) ((\vartheta + \xi)(X_0 - X_p) - \Psi(X_p(t - \varpi), I_p(t - \varpi))) \\ &\quad + \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) ((\xi X_p - \vartheta A_p) - \Lambda(A_p(t - \varpi) I_p(t - \varpi))) \\ &\quad + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\ &\quad + \Lambda(A_p(t - \varpi) I_p(t - \varpi)) - H(I_p(t)). \end{aligned}$$

We now obtain the derivative of $V_2(t)$ as below:

$$\begin{aligned} \frac{dV_2(t)}{dt} &= -\Psi(X_p(t - \varpi), I_p(t - \varpi)) + \Psi(X_p(t), I_p(t)) \\ &\quad - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) + \Lambda(A_p(t), I_p(t)). \end{aligned}$$

Thus, the derivative of $V(t)$ is obtained as:

$$\begin{aligned} \frac{dV(t)}{dt} &= \frac{dV_1(t)}{dt} + \frac{dV_2(t)}{dt} \\ &= \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Psi(X_0, I_p(t))}{\Psi(X_p, I_p(t))}\right) ((\vartheta + \xi)(X_0 - X_p) - \Psi(X_p(t - \varpi), I_p(t - \varpi))) \\ &\quad + \left(1 - \lim_{I_p \rightarrow 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) ((\xi X_p - \vartheta A_p) - \Lambda(A_p(t - \varpi) I_p(t - \varpi))) \\ &\quad + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\ &\quad + \Lambda(A_p(t - \varpi) I_p(t - \varpi)) - H(I_p(t)) - \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\ &\quad + \Psi(X_p(t), I_p(t)) \\ &\quad - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) + \Lambda(A_p(t), I_p(t)). \end{aligned}$$

The postulates P4–P6 imply that

$$\begin{aligned}
 \frac{dV(t)}{dt} &\leq (\vartheta + \xi) \left(1 - \frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} \right) (X_0 - X_p) \\
 &\quad + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} - 1 \right) \\
 &\quad + \left(1 - \frac{\Lambda'_{I_p}(A_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} \right) (\xi X_p - \vartheta A_p) \\
 &\quad + \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Lambda'_{I_p}(A_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} - 1 \right) \\
 &\quad + \frac{I_p(t)}{\vartheta + d + \theta} (R_0 - 1) \\
 &= \vartheta \left(1 - \frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} \right) (X_0 - X_p) + \xi X_0 \left(1 - \frac{X_p}{X_0} \right) \frac{A_0}{A_p} \frac{X_0}{X_p} \left(\frac{X_p}{X_0} - \frac{A_p}{A_0} \right) \\
 &\quad + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Psi'_{I_p}(X_0, 0)}{\Psi'_{I_p}(X_p(t), 0)} - 1 \right) \\
 &\quad + \vartheta (A_0 - A_p) \left(1 - \frac{\Lambda'_{I_p}(A_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} \right) \\
 &\quad + \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Lambda'_{I_p}(A_0, 0)}{\Lambda'_{I_p}(A_p(t), 0)} - 1 \right) + \frac{I_p(t)}{\vartheta + d + \theta} (R_0 - 1).
 \end{aligned}$$

Thus, $R_0 \leq 1$ implies that $\frac{dV(t)}{dt} \leq 0$ for all $t \geq 0$. Also, $\frac{dV(t)}{dt} = 0$ if $X_p(t) = X_0$, $A_p(t) = A_0$, and $I_p(t) = 0$.

Hence, from the system (6), it follows that the largest invariant set $\left\{ (X_p(t), A_p(t), I_p(t) \in R^3_+ \mid \frac{dV(t)}{dt} = 0) \right\}$ is singleton set $\{E_0\}$. Using the Lyapunov-LaSalle asymptotic stability theorem [23, 26, 35], E_0 is the only equilibrium of the system (6) which is globally asymptotically stable. \square

Proof of Theorem 10

Proof For this, the Lyapunov functional is defined as

$$W(t) = W_1(t) + W_2(t),$$

where,

$$\begin{aligned}
 W_1(t) &= X_p(t) - X_p^* - \int_{X_p^*}^{X_p(t)} \frac{\Psi(X_p^*, I_p^*)}{\Psi(\phi, I_p^*)} d\phi + A_p(t) - A_p^* \\
 &\quad - \int_{A_p^*}^{A_p(t)} \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(\Lambda, I_p^*)} d\phi + I_p(t) - I_p^* - I_p^* \ln \frac{I_p(t)}{I_p^*}, \\
 W_2(t) &= \Phi(X_p^*, I_p^*) \int_0^\varpi \left(\frac{\Phi(X_p(t - \theta), I_p(t - \theta))}{\Phi(X_p^*, I_p^*)} - 1 - \ln \frac{\Phi(X_p(t - \theta), I_p(t - \theta))}{\Phi(X_p^*, I_p^*)} \right) d\theta
 \end{aligned}$$

$$+ \Lambda(A_p^*, I_p^*) \int_0^{\varpi} \left(\frac{\Lambda(A_p(t-\theta), I_p(t-\theta))}{\Lambda(A_p^*, I_p^*)} - 1 - \ln \frac{\Lambda(A_p(t-\theta), I_p(t-\theta))}{\Lambda(A_p^*, I_p^*)} \right) d\theta.$$

(P1.)–(P3.) imply that, $W(t) = W_1(t) + W_2(t)$ is defined and continuously differentiable for all $X_p(t)$, $A_p(t)$, $I_p(t) > 0$ and $W(0) = 0$ at $E_c(X_p^*, A_p^*, I_p^*)$.

We compute the derivative of $W_1(t)$ as follows:

$$\begin{aligned} \frac{dW_1(t)}{dt} &= \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) X_p'(t) + \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) A_p'(t) + \left(1 - \frac{I_p^*}{I_p(t)} \right) I_p'(t) \\ &= \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) ((\vartheta + \xi)(X_p^* - X_p(t)) \\ &\quad + \Psi(X_p^*, I_p^*) - \Psi(X_p(t-\varpi), I_p(t-\varpi))) \\ &\quad + \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) (\xi X_p - \vartheta A_p - \Lambda(A_p(t-\varpi), I_p(t-\varpi))) + \left(1 - \frac{I_p^*}{I_p(t)} \right) \\ &\quad \times (\Psi(X_p(t-\varpi), I_p(t-\varpi)) + \Lambda(A_p(t-\varpi), I_p(t-\varpi))) \\ &\quad - \frac{(\Psi(X_p^*, I_p^*) + \Lambda(A_p^*, I_p^*) - h(I_p^*))}{I_p^*} I_p(t) - h(I_p(t)) \\ &= (\vartheta + \xi) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) (X_p^* - X_p(t)) + \Psi(X_p^*, I_p^*) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) \\ &\quad + \frac{\Psi(X_p(t-\varpi), I_p(t-\varpi))}{\Psi(X_p(t), I_p^*)} + (\xi X_p - \vartheta A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\ &\quad - \Lambda(A_p(t-\varpi), I_p(t-\varpi)) \frac{I_p^*}{I_p(t)} \\ &\quad + \frac{\Lambda(A_p(t-\varpi), I_p(t-\varpi))}{\Lambda(A_p(t), I_p^*)} \Lambda(A_p^*, I_p^*) - \frac{I_p^*}{I_p(t)} \Psi(X_p(t-\varpi), I_p(t-\varpi)) \\ &\quad + \Psi(X_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) \\ &\quad + \Lambda(A_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) + \left(h(I_p^*) - \frac{h(I_p(t)I_p^*)}{I_p(t)} \right) \left(\frac{I_p(t)}{I_p^*} - 1 \right). \end{aligned}$$

Since $\xi X_p^* - \vartheta A_p^* - \Lambda(A_p^*, I_p^*) = 0$, or, $-\xi X_p^* + \vartheta A_p^* + \Lambda(A_p^*, I_p^*) = 0$, thus we have

$$\begin{aligned} \frac{dW_1(t)}{dt} &= (\vartheta + \xi) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) (X_p^* - X_p(t)) \\ &\quad + \Psi(X_p^*, I_p^*) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} + \frac{\Psi(X_p(t-\varpi), I_p(t-\varpi))}{\Psi(X_p(t), I_p^*)} \right) \\ &\quad - \frac{I_p^*}{I_p(t)} \Psi(X_p(t-\varpi), I_p(t-\varpi)) + \Psi(X_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) \end{aligned}$$

$$\begin{aligned}
 & + \xi(X_p - X_p^*) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
 & - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \frac{I_p^*}{I_p(t)} + \Lambda(A_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) \\
 & + \vartheta(A_p^* - A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
 & + \Lambda(A_p^*, I_p^*) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p^*)} \right).
 \end{aligned}$$

Now, we compute the derivative of $W_2(t)$ as follows:

$$\begin{aligned}
 \frac{dW_2}{dt} & = \Psi(X_p(t), I_p(t)) - \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\
 & + \Psi(X_p^*, I_p^*) \ln \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \\
 & + \Lambda(A_p(t), I_p(t)) - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \\
 & + \Lambda(A_p^*, I_p^*) \ln \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(X_p(t), I_p(t))}.
 \end{aligned}$$

Thus, the derivative of $W(t)$ with respect to time t is obtained as follows:

$$\begin{aligned}
 \frac{dW}{dt} & = \frac{dW_1}{dt} + \frac{dW_2}{dt} \\
 & = (\vartheta + \xi) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) (X_p^* - X_p(t)) \\
 & + \Psi(X_p^*, I_p^*) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} + \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p^*)} \right) \\
 & - \frac{I_p^*}{I_p(t)} \Psi(X_p(t - \varpi), I_p(t - \varpi)) + \Psi(X_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) \\
 & + \xi(X_p - X_p^*) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
 & - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \frac{I_p^*}{I_p(t)} + \Lambda(A_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} \right) \\
 & + \vartheta(A_p^* - A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
 & + \Lambda(A_p^*, I_p^*) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p^*)} \right)
 \end{aligned}$$

$$\begin{aligned}
& + \Psi(X_p(t), I_p(t)) - \Psi(X_p(t - \varpi), I_p(t - \varpi)) \\
& + \Psi(X_p^*, I_p^*) \ln \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \\
& + \Lambda(A_p(t), I_p(t)) - \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \\
& + \Lambda(A_p^*, I_p^*) \ln \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(X_p(t), I_p(t))} \\
= & \vartheta(X_p^* - X_p) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) + \vartheta(A_p^* - A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
& + \Psi(X_p^*, I_p^*) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} + \ln \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) \\
& + \Psi(X_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} + \ln \frac{I_p}{I_p^*} \right) \\
& + \Lambda(A_p^*, I_p^*) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \ln \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \\
& + \Lambda(A_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*} + \ln \frac{I_p}{I_p^*} \right) \\
& + \Psi(X_p^*, I_p^*) \left(1 - \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \frac{\Psi(X_p(t), I_p^*)}{\Psi(X_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right. \\
& \left. + \ln \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \frac{\Psi(X_p(t), I_p^*)}{\Psi(X_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right) \\
& + \Lambda(A_p^*, I_p^*) \left(1 - \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p(t))} \frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right. \\
& \left. + \ln \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p(t))} \frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right) \\
& + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} - \frac{I_p^*}{I_p(t)} \right) \\
& + \Psi(X_p^*, I_p^*) \left(\frac{\Psi(X_p(t), I_p(t))}{\Psi(X_p^*, I_p^*)} - 1 \right) \\
& + \Lambda(A_p^*, I_p^*) \left(\frac{\Lambda(A_p(t), I_p(t))}{\Lambda(A_p^*, I_p^*)} - 1 \right) \\
& + \Psi(X_p(t - \varpi), I_p(t - \varpi)) \frac{I_p^*}{I_p(t)} \left(\frac{\Psi(X_p(t), I_p^*)}{\Psi(X_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*} \right) \\
& + \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \frac{I_p^*}{I_p(t)} \left(\frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*} \right)
\end{aligned}$$

$$\begin{aligned}
 &+ \Lambda(A_p(t - \varpi), I_p(t - \varpi)) \left(\frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right. \\
 &- \left. \frac{I_p^*}{I_p(t)} \right) + \xi X_p^* \left(1 - \frac{X_p}{X_p^*} \right) \left(\frac{X_p}{X_p^*} - \frac{A_p}{A_p^*} \right) \frac{A_p^* X_p^*}{A_p X_p} \\
 &+ \left(\frac{h(I_p^*)}{h(I_p(t))} - \frac{I_p^*}{I_p(t)} \right) \left(\frac{I_p(t)}{I_p^*} - 1 \right) h(I_p(t)).
 \end{aligned}$$

The functions $\Psi(X_p(t), I_p(t))$ and $\Lambda(A_p(t), I_p(t))$ are monotonically increasing for all $X_p(t) > 0$, and $A_p(t) > 0$. Therefore,

$$\begin{aligned}
 (X_p^* - X_p) \left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) &\leq 0, \\
 (A_p^* - A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) &\leq 0.
 \end{aligned} \tag{39}$$

The function $g(y) = 1 - y + \ln y$, ($y > 0$) has global maximum at $y = 1$. Henceforth, for $y > 0$, $g(y) \leq 0$ and the resulting inequalities are as follows:

$$\begin{aligned}
 &\left(1 - \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} + \ln \frac{\Psi(X_p^*, I_p^*)}{\Psi(X_p(t), I_p^*)} \right) \leq 0, \\
 &\left(1 - \frac{I_p}{I_p^*} + \ln \frac{I_p}{I_p^*} \right) \leq 0, \\
 &\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \ln \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \right) \leq 0, \\
 &\left(1 - \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \frac{\Psi(X_p(t), I_p^*)}{\Psi(X_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right. \\
 &+ \left. \ln \frac{\Psi(X_p(t - \varpi), I_p(t - \varpi))}{\Psi(X_p(t), I_p(t))} \frac{\Psi(X_p(t), I_p^*)}{\Psi(X_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right) \leq 0, \\
 &\left(1 - \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p(t))} \frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right. \\
 &+ \left. \ln \frac{\Lambda(A_p(t - \varpi), I_p(t - \varpi))}{\Lambda(A_p(t), I_p(t))} \frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)} \frac{I_p^*}{I_p(t)} \right) \leq 0.
 \end{aligned} \tag{40}$$

Thus, using (P8.)–(P10.) and the inequalities (39)–(40), it follows that $\frac{dW(t)}{dt} \leq 0$ for all $X_p(t) \geq 0$, $A_p(t) \geq 0$, $I_p(t) \geq 0$. Thus, it is easy to verify that the largest invariant set in $\{(X_p(t), A_p(t), I_p(t)) \in R_+^3 : \frac{dW(t)}{dt} = 0\}$ is singleton $\{E_e\}$. Hence, by the

Lyapunov-LaSalle asymptotic stability theorem [23, 26, 35], the endemic equilibrium E_e is globally asymptotically stable. \square

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