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DYNAMICS OF AN SIR PANDEMIC MODEL USING CONSTRAINED MEDICAL RESOURCES WITH TIME DELAY

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Abstract. The dynamics of the SIR epidemic model are examined in this paper with finite medical resources and variable supply efficiency are examined along with the implications of time delay. This work demonstrates the stability of endemic equilibrium as well as the incidence of backward bifurcation can be significantly impacted by the inclusion of time delay. The theoretical results are supported and supplemented with numerical simulations.

Keywords: SIR epidemic model; variable supply efficiency; time delay; stability of endemic equilibrium; backward bifurcation.

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1. INTRODUCTION

In research on epidemiology, mathematical modeling has become increasingly important in transmission of a communicable illness. Several distinct models for epidemics were proposed

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and thoroughly examined in the research on disease prevention and management [1]. The classic epidemic model typically presupposed that the eradication of infections is inversely correlated with the infective population. This suggests that there are extremely abundant medical resources for infectious diseases, such as medications, antibiotics, hospital wards, and isolation facilities [2],[3],[4],[5] and [6]. The maximum hospital resource availability per unit of time and shows the negative impact of infected patients delaying treatment, which has a substantial effect on the spread of infectious illnesses [7],[8],[9] and [10]. The Chronic illness transmission is significantly impacted by both the lack of medical resources, which is influenced by a variety of factors such as control tactics, drug or vaccine development [11], [12], [13]. In 2004, Wang and Ruan [15], [16] added a constant to SIR model by isolating infectious agents to eliminate the sickness. A constant was used in a SIR model that mimicked a restricted capacity for treatment in order to examine the impact of this limitation on the spread of infectious disease [15] adjusted the ongoing approach to

$$g(I) = \begin{cases} rI^*, & 0 \leq I^* \leq I^*0 \\ rI^*0, & I^* > I^*0 \end{cases} \tag{1}$$

In addition, Wang [16] changed the constant treatment so that the maximum value, rI^*0, was taken when the maximum amount of patients could be treated and the rate of treatment matched the number of infections. The dynamics of various epidemic models with average and standard incidence rates [17], [18] and [19] were investigated by several authors who embraced the staged treatment function. Linhua Zhou and Meng Fan [14] most recently develop a new continuously distinct treatment function in response to the saturation phenomenon of the limited medical resources and meticulously examined the dynamics of the subsequent SIR model.

$$\begin{cases} \frac{dS}{dt} = \Lambda - \frac{\beta SI}{1+KI} - \chi S, \\ \frac{dI}{dt} = \frac{\beta SI}{1+KI} - (\sigma+\gamma+\varepsilon)I - \frac{\alpha I^*}{\omega+I}, \\ \frac{dR}{dt} = \gamma I + \frac{\alpha I}{\omega+I} - \mu R. \end{cases} \tag{2}$$

Here, $\frac{\beta SI}{1+KI}$ is saturated and indicates either the inhibitory effect or the psychological influence. Where $\alpha \geq 0$ denotes the total number of medical assets made available in one time unit and $\omega > 0$ is a constant for part-saturation, which evaluates the efficiency of the availability

of medical assets in that it is more effective if is lower. The dynamics of the SIR model (2) are widely explored in [14] have been thoroughly researched and some intriguing findings, like backward bifurcation and local equilibrium stability, the simulation's dynamics are still far from being finished. We continue to examine the SIR epidemic model's dynamics (2) in [14]. From the aforementioned inspiration study, we have structured our work with delay. We further investigate the Dynamics of a SIR Pandemic Model with Limited Medical Resources based on the earlier arguments.

The proposed time delay model is,

$$\begin{cases} \frac{dS}{dt} = \Lambda - \frac{\beta S(t-\tau)I(t-\tau)}{1+KI(t-\tau)} - \chi s(t), \\ \frac{dI}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{1+KI(t-\tau)} - (\sigma+\gamma+\varepsilon)I(t) - \frac{\alpha I(t-\tau)}{\omega+I(t-\tau)}, \\ \frac{dR}{dt} = \gamma I + \frac{\alpha I}{\omega+I} - \mu R, \end{cases} \tag{3}$$

where, τ represent the time delay. The percentages $S(t)$, $I(t)$, and $R(t)$ correspond to the number of susceptible, infected, and recovered individuals at time t. In section 2, we examine the existence of backward bifurcation and endemic equilibrium as well as the impact of scarce medical resources and the effectiveness of their supply on the backward bifurcation with time delay. We examine the local stability of the equilibria by examining the eigenvalues of the Jacobian matrix. Also, provide a model-wide analysis look at the endemic equilibrium's and the disease-free equilibrium's local and global asymptotic stability. In section 3, The intervention analytical and numerical bifurcation analyses are provided to further the theoretical findings.

2. Backward Bifurcation

The anticipated amount of new sick people that a single diseased person introduces into a population free of illness is known as the basic reproduction number, which is typically denoted by R_0, is one of the essential ideas in dealing with endemic models and is crucial to epidemiology. One frequently notices the threshold property, which states that the disease will not spread if $R_0 < 1$ and will do so if $R_0 > 1$. Forward bifurcation in this context refers to the transition from an endemic equilibrium to equilibrium free of illness. The endemic equilibrium and the disease-free equilibrium coexist when $R_0 < 1$ and the essential criterion for the complete eradication of the disease cannot be the basic reproduction number, as is the case with

TABLE 1. Description of parameters for the system (3)

Symbols	Description
Λ	Recovery rate
χ	Natural death rate of susceptible human population
σ	Natural death rate of infected human population
μ	Natural death rate of recovered human population
β	Transmission rate
γ	Rate at which individuals become infectious after being exposed to the virus
ε	Rate at which individuals lose their immunity to the virus
α	Rate at which individuals are vaccinated or develop immunity through other means
ω	Rate at which individuals are born into the population
t	Time variable
τ	Time delay variable
I	Number of infectious individuals at a given time
K	Effect of social distancing

various models of epidemics that acknowledge backward bifurcation under suitable conditions, according to an increasing number of studies. To get certain essential thresholds for disease control, it is crucial to locate the backward bifurcations. By examining the occurrence of the disease-free and the endemic equilibrium, we explore the feasibility of backward bifurcation in the α-ω plane in this section. According to [20], next generation approach, indicates that the fundamental reproduction number of (3) is,

$$R_0 = \frac{\beta\Lambda}{\chi\left(\sigma+\gamma+\varepsilon+\frac{\alpha}{\omega(t-\tau)+I(t-\tau)}\right)} \tag{4}$$

Similar to the non-delayed model, the delayed model's disease-free equilibrium is described by $E_0 = \left(\frac{\Lambda}{\chi}, 0, 0\right)$.

The algebraic equation below can be solved to produce the endemic equilibrium:

$$\begin{cases} \Lambda - \frac{\beta S(t-\tau)I(t-\tau)}{1+KI(t-\tau)} - \chi s = 0, \\ \frac{\beta S(t-\tau)I(t-\tau)}{1+KI(t-\tau)} - (\sigma+\gamma+\varepsilon)I - \frac{\alpha I(t-\tau)}{1+\omega I(t-\tau)} = 0, \end{cases} \tag{5}$$

which has the following quadratic equation solution:

$$AI^2 + BI + C = 0. \tag{6}$$

Where,

$$\begin{cases} A = (\chi + k\beta)(\sigma+\gamma+\varepsilon) - \omega\alpha\beta, \\ B = -\beta\Lambda + (\chi+k\beta)(\sigma+\gamma+\varepsilon+\omega\alpha) - \frac{d\alpha}{\omega}, \\ C = \alpha\chi - \beta\Lambda\omega + \omega\chi(\sigma+\gamma+\varepsilon). \end{cases}$$

The roots of the quadratic equation can be obtained using the quadratic formula:

$$I_{1,2} = \frac{-B(t) \pm \sqrt{B(t)^2 - 4AC}}{2A}.$$

The corresponding values of susceptible can be obtained using the equation:

$$S_{1,2} = \frac{\Lambda(1+kI_{1,2}(t))}{\chi + k\beta I_{1,2}(t-\tau_0)}.$$

These values of susceptible represent the candidates for the endemic equilibrium of the delayed model.

$$\alpha I^2 + \left[(\beta+\chi k)(\sigma+\gamma+\varepsilon) - \frac{d\alpha}{\omega}\right] I + \alpha\Lambda = 0$$

Where,

$$E_2 = (S_2, I_2),$$

$$S_2 = \left[\Lambda\left[d + (\beta+\chi k)I_2\right]\right]^{-\frac{1}{1+kI_2}},$$

where I_2 is the quadratic equation's bigger root.

Here, Ω_1, Ω_2,and Ω_3 are defined as in the original theorem, and $\alpha_0(\omega)$ is given by:

$$\alpha_0(\omega) = \frac{\omega^2(\beta+\chi k)(\sigma+\gamma+\varepsilon)}{d - \omega(\beta+\chi k)}.$$

Theorem 1. *Assuming $R^* > 1$, we can make the following statements about the equilibrium of this DDE:*

(i) If $R_0 > 1$, then the endemic equilibrium E_2 of DDE is distinct.

(ii) If $P^* < R_0 < 1$ and $(\omega, \alpha) \in \Omega_3$, then DDE of (5) endemic equilibrium E_1 and E_2 exists.

(iii) If $R_0 = 1$ and $(\omega, \alpha) \in \Omega_3$, then DDE of (5) endemic equilibrium E_2 exists.

(iv) If $R_0 = P^*$ and $(\omega, \alpha) \in \Omega_3$, then the endemic equilibrium $\mathrm{E}_1 = \mathrm{E}_2$ of DDE is distinct.

(v) If $0 < R_0 < P^*$ and $(\omega, \alpha) \in \Omega_3$, then there is no endemic equilibrium in DDE of (5).

(vi) If $0 < R_0 \leq 1$ and $(\omega, \alpha) \in \Omega_1 \cup \Omega_2$, then there is no endemic equilibrium in DDE of (5).

Proof. By the model (5) we have:

$$\frac{dN(t)}{dt} = rN(t)\left[1 - \left(\frac{N(t-\tau_1)}{K_1}\right)^{P_1}\right] - \frac{\beta N(t-\tau_2)}{1+\alpha N(t-\tau_3)},$$

disease transmission, and the recovery of infected individuals, respectively. However, the calculations become much more complicated due to the presence of time delays.

In general, the existence of temporal delays can significantly affect the system's dynamics and cause complicated behaviors to emerge in emergency situations.

Let τ be the time delay, then the modified equation becomes,

$$N'(t) = rN\left[1 - \frac{N(t-\tau)}{K}\right] - CN(t).$$

In this equation, N (t) stands for the nation's size at time t, r for intrinsic growth, K for carrying capacity, C for per capita mortality rate, and τ for time delay. Where $N(t-\tau)$ denotes the total number of infected people at time $t-\tau$.

The condition for the existence of an endemic equilibrium E_2 is given by the positive root of equation (6).

Let $D = 1 - \frac{r}{K}N(t-\tau)$ then the modified equation (6) becomes,

$$f(\omega) = -d - \alpha\omega + \frac{\beta(1-e^{-\omega\tau})}{\omega+\gamma+D(t)}.$$

The following prerequisites must be met for endemic equilibrium E_2 to exist in the presence of temporal delay:

(1) If $R_0 < 1$, then it has no endemic equilibrium.

(2) If $R_0 > 1$, then (6) has a unique positive root when $C < 0$ and the condition for the existence of E_2 is given by $P_1 < 1$, where

$$P_1 = 1 + \frac{\varphi(\omega)}{\sigma+\gamma+\varepsilon+\alpha}, \tag{7}$$

and,

$$\varphi(\omega) = (\beta+\chi k)(\sigma+\gamma+\varepsilon)\omega^2 + \alpha\omega(\beta+\chi k) - d + \frac{\beta e^{-\omega\tau}}{\omega+\gamma+\varepsilon+D(t)}. \tag{8}$$

Therefore, in the presence of time delay, the condition for the existence of endemic equilibrium E_2 depends on the value of which in turn depends on the parameters of the system and the time delay τ. We introduce time delay in the model and consider the following delay differential equations:

$$\begin{cases} \frac{dI_1}{dt} = \omega\left[\frac{(1-I_1(t-\alpha))}{N}\right] I_1(t-\alpha) - \frac{\beta I_1(t-\tau)S_1(t-\tau)}{N-I_1(t-\tau)-I_2(t-\tau)} - d_1 I_1^* C, \\ \frac{dI_2}{dt} = \omega\left[\frac{(1-I_2(t-\alpha))}{N}\right] I_2(t-\alpha) - \frac{\beta I_2(t-\tau)S_2(t-\tau)}{N-I_1(t-\tau)-I_2(t-\tau)} - d_2 - I_2^* C, \\ \frac{dS_1}{dt} = \mu N - \mu S_1(t) - \frac{\beta S_1(t-\tau)I_1(t-\tau)}{N-I_1(t-\tau)-I_2(t-\tau)} - \delta_1 S_1(t), \\ \frac{dS_2}{dt} = \mu N - \mu S_2(t) - \frac{\beta S_2(t-\tau)I_2(t-\tau)}{N-I_1(t-\tau)-I_2(t-\tau)} - \delta_2 S_2(t), \end{cases} \tag{9}$$

where τ is time delay, and $N = S_1 + S_2 + I_1 + I_2$ is the total population. The conclusion reached in the absence of time delay still hold in the presence of time delay. We consider the same cases as before:

Case i: $R_0 = P^*, R^* > 1$ and $(\omega, \alpha) \in \Omega_3$.

As the phenomenon, we have $\Delta = 0$, and $I_1(t) = I_2(t)$ for every t. Also, we have $C > 0$ and $B < 0$. So a special endemic equilibrium $E_1 = E_2$ exists in (5), which persists in the presence of time delay.

Case ii: $R^* > 1$ and $(\omega, \alpha) \in \Omega_3$.

In this case, we have $\Delta = 0$ when $P_1 < R_0 < P^*$ and $B \geq 0$ when $0 < R_0 \leq P_1$. Thus, if $R* > 1$ and $0 < R_0 < P^*$and $(\omega, \alpha) \in \Omega_3$, then no endemic equilibrium exists in (5). This conclusion still holds in the presence of time delay.

Case iii: $0 < R_0 \leq 1$ and $(\omega, \alpha) \in \Omega_1 \cup \Omega_2$.

In this case, we have $B \geq 0$ and $C \geq 0$, and no positive root exists for (6). Hence, there is no endemic equilibrium in (5). This conclusion reached in the absence of time delay still hold in the presence of time delay, the evidence is conclusive.

Theorem (1) provides a comprehensive understanding of the endemic equilibrium exists in the presence of time delay. The parameters ω and α play an important part in determining the dynamics of the delayed differential equation (5). Specifically, if $(\omega,\alpha) \in \Omega_1 \cup \Omega_2$, then no endemic equilibrium exists in (5). For $0 < R_0 < 1$ and a distinctive endemic E_2 when $R_0 < 1$. From (5), a forward bifurcation is visible at $R_0 = 1$, where the healthy equilibrium to one single endemic equilibrium E_0 changes.

If $(\omega,\alpha) \in \Omega_3$, then in (5), the endemic equilibrium E_2 is special when $R_0 > 1$. When $P^* < R_0 < 1$, E_1 and E_2 are two separate endemic equilibria in (5), and there is no endemic balance when $0 < R_0 < P^*$. Therefore, (5) displays a reverse bifurcation where E_0 transitions to two parametric equilibria E_1 and E_2. □

Theorem 2. *$R^* > 1$ and $(\omega,\alpha) \in \Omega_3$ with τ if and only if, (5) exhibits a backward bifurcation at $R_0 = 1$. Specifically, if $(\omega,\alpha) \in \Omega_3$, then a special endemic equilibrium E_2 exists in (5), whenever $R_0 > 1$ and the endemic equilibrium types E_1 and E_2 exists, when $P^* < R_0 < 1$ with τ. Moreover, no endemic equilibrium exists in (5) when $0 < R_0 < P^*$ with τ.*

Proof. In particular applications, an endemic backward bifurcation with equilibrium when $R_0 < 1$ is very significant. The actual critical threshold for curing a disease is P^*. The reversible bifurcation from the disease-free equilibrium E_0 at $R_0 = 1$ result in the establishment of both endemic balances E_1 and E_2 for $P^* < R_0 < 1$, as shown in previous theorem with time delay. The phase portrait of (5) is shown in Fig. 1b for $(\omega,\alpha) \in \Omega_3$ and R_0 with time delay, where E_0 is an equilibrium devoid of disease and E_1 and E_2 are two endemic equilibrium states. According to the premise that E_1 refers to a saddle and the fact that E_2 and E_0 are asymptotically stable at the local level proves that a backward bifurcation has taken place. □

2.1. Dynamics on a global scale and the stability of equilibria. Here, we discuss the analysis of the globalized dynamics dynamics of (5) by investigating the regional consistency of equilibrium, which will involve examining the Jacobian matrix eigenvalues for each equilibrium position. Specifically, we will start by considering the (5), Jacobian matrix evaluated at the optimal equilibrium E_0 after a certain time delay.

$$J(E_0) = \begin{bmatrix} -d & -\beta S(t-\tau) \\ 0 & \beta S(t-\tau) - (\sigma+\gamma+\varepsilon+\frac{\alpha}{\omega}) \end{bmatrix},$$

then,

$$\begin{aligned} \det(J(E_0)) &= -d\left[\frac{\beta\Lambda}{d}(t-\tau) - (\sigma+\gamma+\varepsilon+\frac{\alpha}{\omega})\right] \\ &= -d(\sigma+\gamma+\varepsilon+\frac{\alpha}{\omega})[R_0(t-\tau)-1], \end{aligned}$$

and

$$Tr(J(E_0)) = -d + (\sigma+\gamma+\varepsilon+\frac{\alpha}{\omega})[R_0(t-\tau)-1].$$

Theorem 3. *If $R^* \leq 1$ and $(\omega,\alpha) \in \Omega_1 \cup \Omega_2$, then the endemic balance E_1 of (5) is locally unstable if $D > 0$ and locally symmetrically stable if $D < 0$. If $R^* > 1$ and $(\omega,\alpha) \in \Omega_3$, then E_1 is locally symmetrically stable, if $R_0 < P_1$ and erratic if $R_0 > P_1$. If $R_0 > 1$ and $R^* > 1$, then E_1 is erratic. If E_1 exists, it is saddle.*

Proof. To prove the theorem, we first consider the system (5), Jacobian matrix evaluated at endemic equilibrium E_1:

$$J(E_1) = \begin{bmatrix} \alpha - \delta(1-\omega I(E_1))/N & 0 \\ 0 & -\gamma-\delta+\delta\omega I(E_1)/N-\mu-\alpha \end{bmatrix},$$

where $I(E_1)$ is the value of I at the endemic equilibriumE_1, and we have used the notation $I(E_1)$ to indicate their dependence. To analysis the stability of E_1, we can use the characteristic equation of $J(E_1)$, which is a third-order polynomial of the form:

$$\Lambda^3 + a\Lambda^2 + b\Lambda + c = 0,$$

where,

$$\begin{aligned} a &= Tr(J(E_1)), \\ b &= -(Tr(J(E_1))^2 - 2\det(J(E_1))), \\ c &= -\det(J(E_1)). \end{aligned}$$

The unique equation has negative roots and zero real components. This requirement can only be met if and only if all roots have a negative real component. In other words, every root must

be in the left side of the complex plane. As a result, the first requirement for stability says that all of the coefficients in the characteristic equation must be positive.

At $\tau = 0$, the endemic equilibrium, E_1, is locally asymptotically stable. This criteria indicates that the intrinsic equilibrium E_1 is locally asymptotically stable in the absence of a temporal delay, which may be confirmed by looking at the Jacobian matrix $J(E_1)$ assessed at $\tau = 0$. Hence, E_1 is a saddle point. □

Theorem 4. *If $R^* > 1$ and $(\omega, \alpha) \in \Omega_3$ are true, the endemic equilibrium E_1 is present.*

Proof. To explore we introduce the presence of time delay τ in the equation for the infected individuals:

$$\frac{dI_2(t)}{dt} = \beta[S(t-\tau) - S(t)]I_2(t) - (\sigma+\gamma+\varepsilon)I_2(t) + \alpha I_1(t-\tau),$$

which leads to the following delayed system.

$$\frac{dS(t)}{dt} = \omega(N - S(t) - I_1(t) - I_2(t)) - \beta S(t)(I_1(t) + I_2(t)),$$

then,

$$\frac{dI_1(t)}{dt} = \beta S(t-\tau)I_1(t-\tau) - (\sigma+\gamma+\varepsilon)I_1(t) - \alpha I_2(t),$$
$$\frac{dI_2(t)}{dt} = \beta S(t-\tau)I_2(t-\tau) - (\sigma+\gamma+\varepsilon)I_2(t) - \alpha I_1(t-\tau).$$

It is trivial to demonstrate that the Jacobian matrix E_2 exists. The Jacobian matrix $J(E_2)$ of this delayed system evaluated at the endemic equilibrium E_2, which is defined below:

$$J(E_2) = \begin{bmatrix} -\omega\beta - \omega(\beta + dk) - \alpha(\beta + \chi k) - \omega\alpha & 0 \\ \alpha\omega & -(\sigma+\gamma+\varepsilon) - \beta - \omega\beta - \alpha(\beta+\chi k) \end{bmatrix}.$$

Next, We determine $J(E_2)$'s characteristic equation, which is given by:

$$\det(SI - J(E_2)) = S^3 + m_1 S^2 + m_2 S + m_3 = 0,$$

where,

$m_1 = \omega(\beta + \chi k + \gamma + \varepsilon + \alpha) + d,$

$m_2 = A_2\omega^2 d - rAC + qBC + (\beta + \chi k)(\sigma + \gamma + \varepsilon + \alpha)\omega^2,$

$m_3 = \omega\beta(\beta + \chi k)(\sigma + \gamma + \varepsilon) + \alpha\omega^2(\sigma + \gamma + \varepsilon) + \alpha(\beta + \chi k)(\sigma + \gamma + \varepsilon) + (\sigma + \gamma + \varepsilon)(\beta + \chi k + \alpha\omega).$

Conditions that must be met in order for E_2 to be locally asymptotically stable are that $m_1 > 0$, $m_2 > 0$, and $m_3 > 0$. However, the conditions for m_1 and m_2 are complicated expressions.

We begin the linearizing the system about E_2:

$$\frac{dS}{dt} = -\mu S + \Lambda_{11} S\tau_1 - \Lambda_{12} I_1 \tau_2 - \Lambda_{13} I_2 \tau_3.$$

Then,

$$\frac{dI_1}{dt} = \Lambda_{21} S\tau_1 - \Lambda_{22} I_1 \tau_2,$$
$$\frac{dI_2}{dt} = -VI_2 + \Lambda_{33} I_1 \tau_3,$$

where $\Lambda_{11} = \beta S_2, \Lambda_{12} = \beta I_2, \Lambda_{13} = 0, \Lambda_{21} = -\beta S_2, \Lambda_{22} = (\beta + dk) I_2, \Lambda_{33} = \gamma$, and,

$$\mu = V + \alpha I_2 + \omega,$$

using time delay, we write the system as follows:

$$\frac{dS(t)}{dt} = -\mu S(t) + \Lambda_{11} S(t-\tau_1) - \Lambda_{21} I_1 (t-\tau_2) - \Lambda_{13} I_2 (t-\tau_3).$$

Therefore,

$$\frac{dI_1(t)}{dt} = \Lambda_{21} S(t-\tau_1) - \Lambda_{22} I_1 (t-\tau_2),$$
$$\frac{dI_2(t)}{dt} = -VI_2(t) + \Lambda_{33} I_1 (t-\tau_3).$$

To obtain the characteristic equation, we assume that the solution of the form, e^{rt}, $e^{\Lambda_1 S_1}, e^{\Lambda_2 S_2}, e^{\Lambda_3 S_3}$ are valid. Here, Λ_i are the Laplace transform of delay functions and S_i are the roots of characteristic equation. We substitute these expressions into the linearized system and obtained:

$$r + \mu + \Lambda_{11} e^{-r\tau_1} \Lambda_1 + \Lambda_{12} e^{-r\tau_2} \Lambda_2 + \Lambda_{13} e^{-r\tau_3} = 0,$$

$$\Lambda_{21} e^{-r\tau_1} \Lambda_1 + r + \Lambda_{22} e^{-r\tau_2} \Lambda_2 = 0,$$

$$V + \Lambda_{33} e^{-r\tau_3} \Lambda_3 + r = 0.$$

By solving r, the determinant of the coefficients of the system is given by

$$\det(J(E_2)) = r^3 + a_2 r^2 + a_1 r + a_0 = 0,$$

where,

$$a_2 = \Lambda_{11}\Lambda_{22} + \Lambda_{11}\Lambda_{33}e^{-\Lambda_3\tau_3} + \Lambda_{22}\Lambda_{33}e^{-\Lambda_2\tau_2},$$

$$a_1 = \Lambda_{11}\Lambda_{22}\Lambda_{33} + \Lambda_{11}\Lambda_3 e^{-\Lambda_3\tau_3} + \Lambda_{11}\Lambda_{22}e^{-\Lambda_2\tau_2}\Lambda_{33}$$
$$+ \Lambda_{22}e^{-\Lambda_2\tau_2}\Lambda_{22}\Lambda_{33} + \mu\Lambda_{11} + V\Lambda_{22} + \mu\Lambda_{33}e^{-\Lambda_3\tau_3},$$

$$a_0 = \mu\Lambda_{22}\Lambda_{33} + V\Lambda_{11}\Lambda_{33} + \mu\Lambda_{11}\Lambda_{33}e^{-\Lambda_3\tau_3} + \mu\Lambda_{22}\Lambda_{33}e^{-\Lambda_2\tau_2} + V\Lambda_{11}\Lambda_{22}e^{-\Lambda_2\tau_2}.$$

Since, E_2 is a positive constant and the other terms are also positive. □

Theorem 5. *E_2 is asymptotically stable when $\mu > 0$ and E_2 is unsteady when $\mu < 0$.*

Proof. We will assume that the conditions for the existence of E_2 are satisfied.

First, we linearize the system around E_2 and obtain the Jacobian matrix:

$$J(E_2) = \begin{bmatrix} -\sigma+\gamma+\varepsilon-\frac{\beta I_2}{(1+kI_2)} & \frac{\beta I_2}{(1+kI_2)} \\ 0 & -\sigma-\gamma-\varepsilon-\delta-\frac{\alpha\omega}{(\omega+I_2)^2} \end{bmatrix},$$

where δ is the time delay. The characteristic equation of $J(E_2)$ is given by

$$\Lambda^3 + a\Lambda^2 + b\Lambda + c = 0$$

where,

$$a = \sigma+\gamma+\varepsilon+\delta-\beta S_2 - \frac{\beta I_2(1+kI_2)}{(1+kI_2)\omega},$$
$$b = \beta S_2\delta + \frac{\beta I_2(1+kI_2)\delta}{(1+kI_2)\omega} - \frac{\sigma+\gamma+\varepsilon+\delta}{(\omega+I_2)^2} - \frac{\beta S_2(d+\gamma+\varepsilon)}{\omega} - \frac{\beta I_2(1+kI_2)(\sigma+\gamma+\varepsilon+\delta)}{\omega(1+kI_2)},$$
$$c = \frac{(\sigma+\gamma+\varepsilon+\delta)\alpha\beta S_2}{(\omega+I_2)^2\omega} - \frac{\beta I_2(1+kI_2)\alpha(\sigma+\gamma+\varepsilon+\delta)}{(1+kI_2)(\omega+I_2)^2\omega}.$$

According to this criterion, E_2 is asymptotically stable if all the coefficient of the characteristic equation is positive, and there are no sign changes in the sequence of the coefficients. Let's define the following expressions: $P^1 = a, P^2 = b - \frac{ac}{3}, P^3 = \frac{c}{3}, q^1 = \frac{P^2}{P^1}, q^2 = \frac{P^1P^3-(P^2)^2}{(P^1)^2}$. Then, the condition for stability is $P^1, P^2, P^3, q^1, q^2 > 0$. Now,

$$P^1 = (\sigma+\gamma+\varepsilon+\delta)(1-R_0),$$

$$P^2 = \delta(\beta S_2 - R_0\omega) - \alpha(\omega+I_2)^2 + \frac{\beta S_2(\sigma+\gamma+\varepsilon)}{\omega} + \frac{\beta I_2(1+kI_2)(R_0\omega-\sigma-\gamma+\varepsilon-\delta)}{\omega(1+kI_2)},$$

$$P^3 = \frac{\beta S_2 \alpha(\sigma+\gamma+\varepsilon+\delta)}{\omega(\omega+I_2)^2} + \frac{\beta I_2(1+kI_2)\alpha(\sigma+\gamma+\varepsilon+\delta)}{\omega(1+kI_2)(\omega+I_2)^2},$$

$$q^1 = \delta(\beta S_2 - R_0\omega) - \alpha(\omega+I_2)^2 + \frac{\beta S_2(\sigma+\gamma+\varepsilon)}{\omega} + \frac{\beta I_2(1+kI_2)(R_0\omega-\sigma-\gamma+\varepsilon-\delta)}{\omega}.$$

If all the eigenvalues are negative, then the endemic equilibrium E_2 is locally asymptotically stable. If any real components of eigenvalue are positive then E_2 is unstable. Since, E_2 is locally asymptotically stable at $\mu > 0$, and E_2 is unstable at $\mu < 0$. □

Theorem 6. *The system (5) contains no closed orbits.*

Proof. Assume by contradiction that there exists a closed orbit in the phase space of system (5), this indicates that a periodic solution exists with period $T > 0$. Without loss of generality, we consider the orbit lies in the first quadrant of the phase space.

Let us construct a suitable Dulac function that satisfies the conditions of the Bendixson-Dulac theorem. Let:

$$D(x,y) = \frac{y}{\omega(x+1)}$$

Then,

$$\frac{\partial}{\partial x}(Df_1) + \frac{\partial}{\partial y}(Df_2) \quad = -\frac{y^2}{\omega(x+1)^3}\left[\frac{\beta x(1+kx)}{(1+kx)^2} - \frac{\delta(\omega+x)}{\omega+y}\right].$$

We have,

$$\begin{aligned}\left(\frac{\beta x(1+kx)}{(1+kx)} - \frac{\delta(\omega+x)}{\omega+y}\right) &\leq \frac{\beta x(1+kx)}{(1+kx)^2} - \delta - \frac{\delta x}{\omega},\\ &\leq \frac{\beta x(1+kx)}{(1+kx)^2} - \delta - \frac{\delta}{k},\\ &= \frac{\beta x - \delta k - \delta kx}{k(1+kx)^2},\\ &\leq 0.\end{aligned}$$

Therefore, for all (x,y), $\frac{\partial}{\partial x}(D(f_1)) + \frac{\partial}{\partial y}(D(f_2))$ lies in the first quadrant.

Since, $\frac{\partial D}{\partial x}$ and $\frac{\partial D}{\partial y}$ are both continuous on the first quadrant, $h(x,y)$ is a continuously differentiable function that occurs in such a way that,

$$\frac{\partial}{\partial x}(h\frac{\partial D}{\partial x}) + \frac{\partial}{\partial y}(h\frac{\partial D}{\partial y}) = h\left(\frac{\partial^2 D}{\partial x^2} + \frac{\partial^2 D}{\partial y^2}\right) + 2\frac{\partial h}{\partial x}\frac{\partial D}{\partial x} + 2\frac{\partial h}{\partial y}\frac{\partial D}{\partial y} \neq 0.$$

Thus, by the Bendixson-Dulac theorem [21], there are no closed orbits in the phase space of system (5). □

Theorem 7. *If* $R_0 > 1, \alpha \leq \omega^2(\beta + \chi k) + \omega^2 k(\sigma + \gamma + \varepsilon) + \omega(2d + \alpha k)$ *then,* E_2 *is globally asymptotically stable.*

Proof. To prove the global asymptotic stability of E_2, we use the Lyapunov function approach. Consider,

$$V(I(t), S(t)) = \omega I^2(t) + S^2(t).$$

Taking the time derivative of V,

$$\frac{dV}{dt} = 2\omega I(t)(I(t) - I^*) - \alpha S(t)I(t) - \beta S(t)I^2(t).$$

Using the expression for I^* from Theorem 7, we can simplify the above expression as

$$\frac{dV}{dt} = -\alpha S(t)I(t) - \beta S(t)(I(t) - I^*)^2 - \mu S^2(t),$$

where, $\mu = \frac{2A(m_2\omega^2 + Bm_2 + m_1)}{\omega}$. Note that $\mu > 0$ for the given conditions on R_0 and α. Therefore, $\frac{dV}{dt}$ is negative definite and $(I(t) - I^*)(I(t), S(t))$ is a Lyapunov function for the system. This suggests that the system's entire set of solutions is covered to the set $I = I^*, S = 0$ as $t \to \infty$.

Since, I^* is the only positive solution making it unique to the equation $\psi(I) = 0$, the only equilibrium in this set is E_2. Hence the proof. □

Theorem 8. *The disease-free equilibrium* E_0 *is globally asymptotically stable if one of the following conditions are satisfied.*

(i) $0 < R_0 < 1, (\omega, \alpha) \in \Omega_1 \cap \Omega_2$

(ii) $0 < R_0 < P^*, (\omega, \alpha) \in \Omega_3$

Proof. From the system (5),

$$\frac{dS}{dt} = \Lambda - (\beta + \delta + \omega)S + \alpha SI(t - \tau)$$
$$\frac{dI}{dt} = \alpha SI(t - \tau) - (\mu + \gamma + \omega)I$$

Case (i): $0 < R_0 < 1, (\omega, \alpha) \in \Omega_1 \cap \Omega_2$

In this case, the ideal state of equilibrium E_0 is the solitary equilibrium point of the system. It

is simple to demonstrate that E_0 is locally stable using the Routh-Hurwitz criteria. Furthermore, the region $D = ((S,I), I \geq 0, S+I \geq 0)$ is positively invariant with regard to the integrity of the system (5). This means that every solution trajectory, a closed orbit or equilibrium that is located in D. Since, condition (i) is holds; No endemic equilibrium exists in the system (5), and hence, there is no closed orbit in D. Since, every solution trajectory beginning in D will therefore go closer to the disease-free equilibrium E_0. By the Poincare-Bendixson theory, E_0 is asymptotically stable everywhere. The evidence for case (i) is now complete.

Case (ii): $0 < R_0 < P^*, (\omega, \alpha) \in \Omega_3$

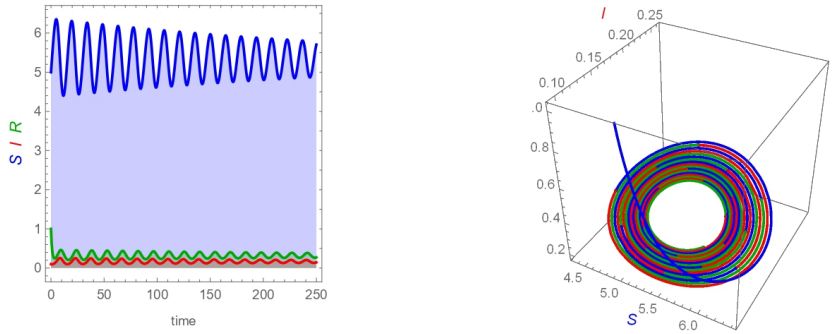
In this case, there exists at least one endemic equilibrium E^* of the system (5). It is simple to demonstrate that E^* is locally stable using the Routh-Hurwitz criteria. Furthermore, with regard to the system, the region known as D is still positively invariant (5). Since, condition (ii) is holds; the system (5) has no closed orbits in D. Therefore, every solution trajectory starting in D will approach either the equilibrium without disease E_0 or the epidemic equilibrium E^*. By the Lasalle invariance principle [22], every solution trajectory starting in D will approach the largest invariant set contained in the union of the unaffected equilibrium E_0 and the infectious equilibrium E^*. If the initial condition $(S(0), I(0))$ in D, then $S(t) \geq 0$ for all $t \geq 0$. Since, $R_0 < P^*$, the endemic equilibrium E^* is unstable. Therefore, each solution trajectory will move closer to the disease-free equilibrium E_0 as $t \to \infty$ starting from D. By the Poincare-Bendixson theory, E_0 is asymptotically stable. The evidence for case (ii) is now complete. Therefore, we have shown that if either condition (i) or condition (ii) is satisfied. Consequently, the disease free equilibrium E_0 is globally asymptotically stable. □

3. NUMERICAL SIMULATION

A numerical simulation is used to examine how infectious illnesses spread throughout a population. According to this theoretical framework, there are three groups of people in society: those who are vulnerable to catching the disease, those who are infectious and may spread it, and those who have recovered from the sickness or who have already passed away from it. The starting population of susceptible and infected people, as well as the assumption of a certain transmission rate and recovery rate, all form the basis of the simulation. The simulation then

runs in discrete time steps, updating the population of each compartment according to the established equations.

Fig.1. Shows that the infected and healed populations eventually die extinct in the situation we described, however, shows that the illness does not last or is unable to create long-lasting illnesses within the community. The existence of external variables that regularly impact the population's sensitivity to the disease may be the cause of the periodic oscillation in the susceptible population. This might be caused by a number of factors, including efficient control methods, quick and widespread immunization, or the pathogen's inability to survive for an extended amount of time inside the host population.



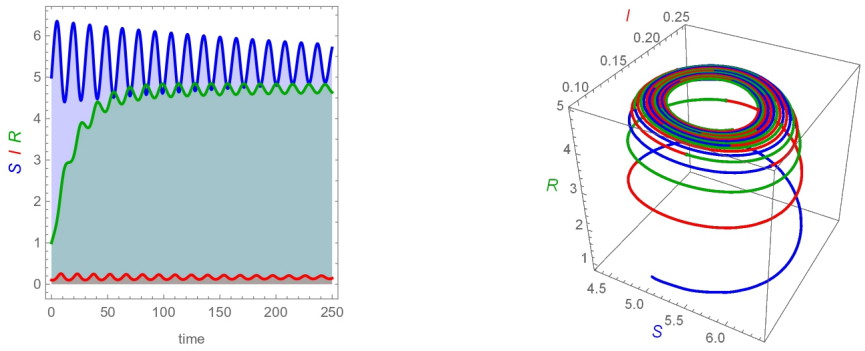
(A) (B)

FIGURE 1. Periodic oscillation in SIR population

Fig.1. In this figure, the ratio of the infective size at equilibrium to R_0 vary by 0.01 to 1.5, correspondingly, R_0 increases in susceptible by higher ratio. From the disease-free equilibrium, there occurs a forward bifurcation by $R_0 > 1$ at $R_0 = 1$, resulting in the establishment of a special endemic equilibrium E_2 $(R_0 < 1)$. Here, $\chi = 0.01$, $\alpha = 1$, $\Lambda = 1.5$, $\mu = 1$, $\beta = 1$, $\omega = 1$, $\gamma = 1$, $\sigma = 1$, $\varepsilon = 1$, $\tau = 0.01$.

In Fig.2. The susceptible population may occasionally come into touch with the pathogen, which might result in reinfection, due to a variety of reasons, including the infectious agent's dynamics of transmission. As a result, vulnerable and immune populations fluctuate. Eventually, the affected population becomes extinct. This could happen if the disease's transmission rate falls off sharply over time or if other elements, such successful public health initiatives,

restrict the disease's spread. The affected population may therefore decrease until it approaches zero.



(A) (B)

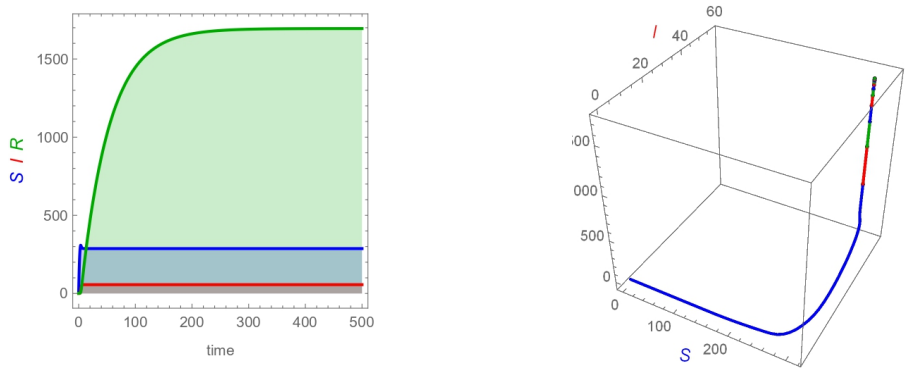
FIGURE 2. Periodic oscillation in dynamics of transmission

Fig.2. In this figure, the ratio of the infective size at equilibrium to R_0 vary by 0.01 to 1.25, correspondingly, R_0 increases in recovered by higher ratio. From the disease-free equilibrium, there occurs a backward bifurcation at $R_0 = 1$, ends up resulting in a variety of endemic equilibrium states $P^* < R_0 < 1$. Here, $\chi = 0.01$, $\alpha = 1$, $\Lambda = 1.25$, $\mu = 1$, $\beta = 1$, $\omega = 1$, $\gamma = 1$, $\sigma = 1$, $\varepsilon = 0.03$, $\tau = 0.01$.

Fig.3. Describes the population consists of both susceptible people (who are still at danger of catching the illness) and recovered people (who have developed immunity and are shielded against reinfection). If the pathogen comes into touch with the vulnerable people, they might get infected. Despite the existence of vulnerable people, the population of those who are infected is gradually getting less.

Fig.3. Coexistence of susceptible and recovered individuals and phase portrait of (3) also included, where there are disease-free equilibrium at E_0 and dual endemic balances E_1 and E_2. E_2 and E_0 are locally asymptotically stable, whereas E_1 is a saddle. Here, $\chi = 0.50$, $\alpha = 0.10$, $\Lambda = 200.0$, $\mu = 0.02$, $\beta = 0.20$, $\omega = 0.25$, $\gamma = 0.6$, $\sigma = 0.1$, $\varepsilon = 0.30$, $\tau = 0.01$. Whereas, $(\omega, \alpha) \in \Omega_1 \cap \Omega_2$ and the backward bifurcation occurs.

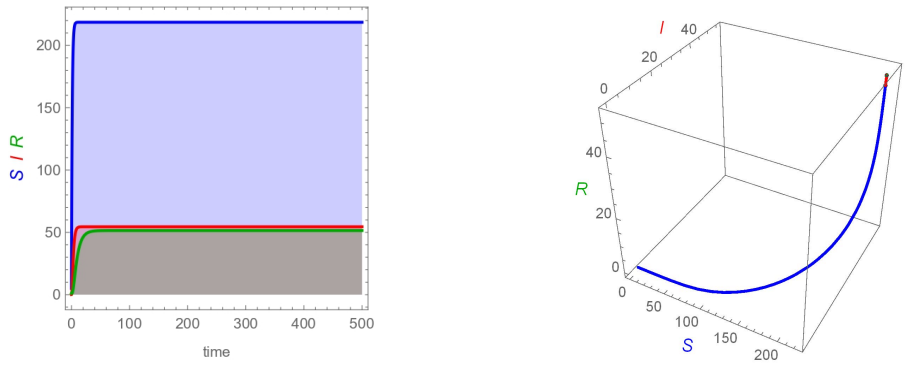
Fig.4. Illustrates the coexistence of these three populations in the SIR model illustrates the



(A) (B)

FIGURE 3. Coexistence of SIR individuals at E_0 with $\Lambda = 200.0$ and $\beta = 0.20$.

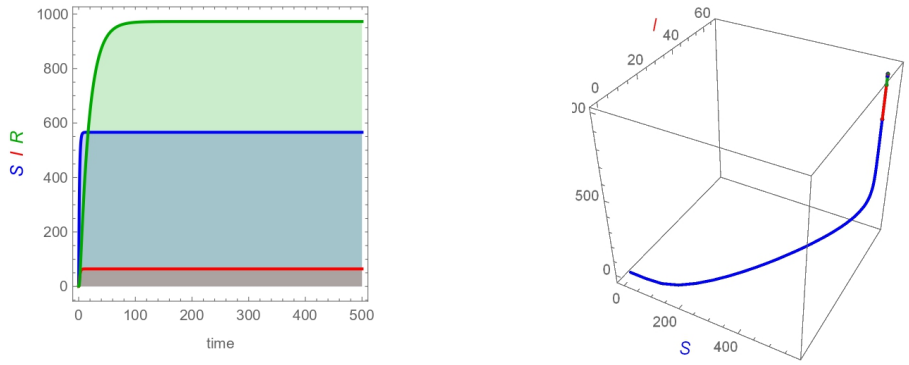
continues interplay between susceptible individuals becoming infected, infected individuals recovering or succumbing to the disease, and recovered individuals building immunity. The model captures the complex dynamics of disease transmission and highlights the importance of factors such as population susceptibility, the infectiousness of the disease, and the development of immunity in shaping the overall course of the epidemic.



(A) (B)

FIGURE 4. Coexistence of SIR individuals at E_0 with $\Lambda = 170.0$ and $\beta = 0.13$.

Fig.4. In this figure both the ratio and the Phase portrait of (3) also included and the endemic equilibrium is locally asymptotically stable whereas the disease-free equilibrium, E_1 is unstable. Here, $\chi = 0.65$, $\alpha = 0.12$, $\Lambda = 170.0$, $\mu = 0.14$, $\beta = 0.13$, $\omega = 0.22$, $\gamma = 0.13$, $\sigma = 0.14$, $\varepsilon = 0.24$, $\tau = 0.01$. Whereas, $(\omega, \alpha) \in \Omega_3$ and the backward bifurcation occurs.



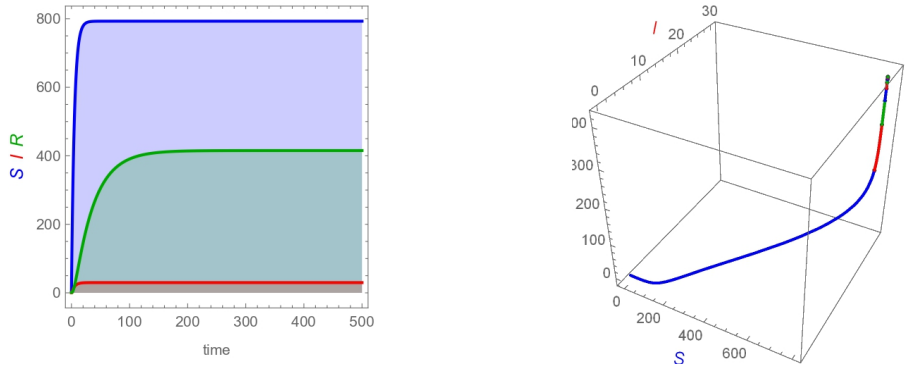
(A) (B)

FIGURE 5. Coexistence of SIR individuals at E_0 with $\Lambda = 400.0$ and $\beta = 0.21$.

Fig.5. To examine our model's dynamical behaviour the system (3) is simulated and included, Similarly, it is straightforward to see that the R_0 value rises in these parameters, E_1 and E_2. It is simple to see that if we desire a stable, disease-free equilibrium, we must need to increase our detection parameters. Here, $\chi = 0.500$, $\alpha = 0.3$, $\Lambda = 400.0$, $\mu = 0.06$, $\beta = 0.21$, $\omega = 0.26$, $\gamma = 0.9$, $\sigma = 0.6$, $\varepsilon = 0.31$, $\tau = 0.01$. Whereas, the system (3) has two possible equilibrium states, one of which is endemic and the other is devoid of illness.

Fig.6. Illustrates how a sizeable section of the population has either developed immunity or made a full recovery, which has decreased the number of people who are vulnerable to the disease and broken the chain of transmission. This result, which emphasizes the need of preventative measures, early identification, efficient treatments, and interventions to eradicate or minimize the burden of infectious illnesses on a community, is frequently the desired outcome in public health initiatives.

Fig.6. Bifurcation surface of ω and α (Variations in the dynamics of the infected population's size ω and α) which illustrate the influence of and on the spread of illnesses. [a] $(\omega, \alpha) \in \Omega_1 \cap \Omega_2$, [b] $(\omega, \alpha) \in \Omega_3$. It demonstrates that more medical resource sufficiency (larger α) and greater medical resource supply efficiency (smaller ω) might decrease the size of the infective population. Here, $\chi = 0.1$, $\alpha = 0.51$, $\Lambda = 156$, $\mu = 0.03$, $\beta = 0.1$, $\omega = 0.5$, $\gamma = 1.4$, $\sigma = 0.65$, $\varepsilon = 0.5$, $\tau = 0.01$. Whereas, $(\omega, \alpha) \in \Omega_1 \cap \Omega_2$ and the backward bifurcation occurs.



(A) (B)

FIGURE 6. Coexistence of SIR individuals at E_0 with $\Lambda = 156$ and $\beta = 0.1$.

4. CONCLUSION

We investigated the dynamics of SIR epidemic model of supply variability in time delay and resource constraints on availability of medical care. We demonstrated the increase in temporal delay can have a considerable impact on both the occurrence of backward bifurcation and the beginning and stability of endemic equilibrium. The SIR compartmental model is also obtained from the value of balanced infectious sizes versus R_0. Finally, our theoretical conclusions were strengthened and verified by numerical simulations for the system with a time delay.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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