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THE ROLE OF SATURATED INCIDENCE AND TREATMENT RATES ON THE DYNAMICS OF EPIDEMIOLOGICAL MODEL

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Abstract: This paper examines an epidemic model of the SEIR type that was created to account for the impact of immunization campaigns and saturated therapy. To determine the effects of inadequate health care and the efficacy of vaccinations in halting the spread of infection, the model was mathematically examined. Both local and global stability were examined, and equilibrium points were computed. Additionally, the pattern may exhibit inverse branching, according to the research, suggesting that the sickness can persist even when $R_0 < 1$. A sensitivity study was used to determine which aspects of the proliferation dynamic were most crucial. Numerical findings corroborate theoretical predictions and highlight the need to combine medication and vaccination as effective epidemic management strategies.

Keywords: vaccination; saturated treatment; basic reproduction number.

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1. INTRODUCTION

Since mathematical models can explain the interaction between exposed and infected individuals within a given community, researchers are increasingly interested in using them to understand the dynamics of disease transmission as a result of the rising prevalence of infectious

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diseases. Researchers have used a variety of models to assess health interventions and identify the most effective strategy to manage them; for instance, refer to [1-3]. Classical models like the SIR model, which was introduced by Kermack and McKendrick [4] in 1927 and postulated infinite treatment and linear transmission, served as the foundation for this investigation. The reader can view these studies [5-7] in addition to the controlled treatment. Then, a number of improved models that consider the rates of saturated infection [8-10] emerged using a function $\frac{\beta SI}{1+bI}$, where this rate reflects the change in behavior of individuals when the number of infected increases, where b is the level of saturation.

Currently, researchers have proposed a number of disease control models that consider vaccine and treatment, two crucial and successful strategies to slow the spread of infection. For instance, in their models, the researchers only included therapy [11-12]. Other people utilized the vaccine [13-14], and other people mixed the two, but they took the treatment in a linear fashion [15-17]. Zhang and Liu recently took the nonlinear (saturated) treatment function in 2008 [18], presenting it as $T(u,I) = \frac{\phi uI}{1+\alpha uI}$, which is taken here as a function of both the control u and the infected individual I. It has been observed that the treatment function likewise tends to a value $T(u,I) \approx \phi uI$ at very low values of I or u, and to $T(u,I) \approx \frac{\phi}{u}$ at very large values. This only uses a continuous and differentiable function to describe the saturation feature of the treatment. Furthermore, the opposite effect of delaying treatment for the infected is described by $\frac{1}{1+\alpha uI}$. Ultimately, the saturated treatment function reverts to the linear one at $u = 0$. These models demonstrate that some patients may postpone treatment if the number of infections rises, which could aid in the disease's ongoing spread. This is because the amount of treatment is described by a nonlinear (saturated) function. For more accurate results, it is crucial to incorporate this kind of function into epidemiological models like SEIR, for instance [19-21].

However, vaccination is crucial when it comes to being included in mathematical models [22-24]. In order to reduce the number of people exposed to harm and its direct impact on the fundamental reproduction number (R_0), vaccination is essential. The SEIR model was widely used in the study of epidemiological models of the spread of infectious diseases that have an incubation period, such as COVID-19, measles, and influenza A (H1N1); see [25-27]. Based on the aforementioned, the current study puts forward an epidemic model of the SEIR type improved

from the research [19] that involves the rate of saturated infection and saturated therapy, in addition to incorporating the impact of vaccination campaigns on disease control. Apart from applying the virus spread model, which is the best at comprehending the connections between the following categories: susceptible (S), exposed (E), infected (I), and recovered persons (R). Newborns and those just joining the community will receive vaccinations; the fraction of vaccinated persons is represented by the extra parameter θ, where $0 < \theta < 1$, see [28]. Regarding treatment, a suitable dosage will be given, with the amount given being carefully regulated. Thus, combining these elements into a single model gives us a more accurate representation of the relationship between prevention and treatment, which aids in the creation of a health strategy that strikes a balance between the two.

The following is how this document is structured. The model's mathematical formulation with the solution's attributes is described in the following section. The basic reproduction number and equilibrium points are established in Section 3. In Section 4, the model's local stability analysis is examined. The model's global stability is covered in Section 5. Section 6 examines the presence and bifurcations of equilibria for a model that incorporates backward bifurcation. The sensitivity analysis is examined in Section 7. To validate our findings, we provide a few numerical simulations in Section 8. Lastly, Section 9 offers a succinct conclusion.

2. MODEL FORMULATION

In this part, an epidemiological model of the SEIR type is simulated mathematically by constructing a mathematical model. Let $N(t)$ be the total population size at time t, which divides into four categories: the susceptible $S(t)$, the exposed $E(t)$, the infected $I(t)$, and the recovered $R(t)$. The diseases are transmitted to the susceptible $S(t)$ group by contact with infected people $I(t)$. The health prevention is considered by giving the population from the category of vulnerable to infection a vaccine, where the effect of the preventive vaccine can be explained by the way some people were vaccinated at birth, and the number of recoveries $R(t)$ can be increased by saturated treatment, and thus the sports model can be described through the following equation

$$\begin{aligned}
\frac{dS}{dt} &= (1-\theta)\Omega - \frac{\beta SI}{1+bI} - \mu S + \psi R = g_1(S,E,I,R),\\
\frac{dE}{dt} &= \frac{\beta SI}{1+bI} - \mu E - \delta E = g_2(S,E,I,R),\\
\frac{dI}{dt} &= \delta E - \frac{\phi uI}{1+\alpha uI} - \mu I - \rho I - \gamma I = g_3(S,E,I,R),\\
\frac{dR}{dt} &= \theta\Omega + \frac{\phi uI}{1+\alpha uI} + \rho I - \mu R - \psi R = g_4(S,E,I,R).
\end{aligned} \tag{1}$$

All the parameters in the equation are non-negative and can be described in detail as shown in Table 1. It is assumed that the susceptible population is at risk of injury when there is direct contact with the infected population, according to the saturated incidence rate $\frac{\beta SI}{1+bI}$. However, the infection is reduced by using treatment according to the saturated function $\frac{\phi uI}{1+\alpha uI}$, or by the body's defense with a rate of ρ. The recovered individuals may lose their immunity at a rate ψ. The transition from the exposed category to the infected category is done at a rate δ. Finally, all the populations face a natural death at a rate μ.

Table 1. Parameters Description.

Parameters	Description
θ	Represents the rate of people vaccinated at birth.
Ω	The recruitment rate
β	Infection prevalence coefficient (Transmission rate).
b	Inhibition of the incidence growth rate at high I (Saturation factor).
μ	Natural death rate.
ψ	The probability of losing immunity.
δ	The infection rate.
u	The treatment control function
ϕ	Treatment effectiveness or maximum energy therapy function.
α	The saturation of the treatment level.
ρ	Recovery rate from the disease.
γ	The death rate caused by the disease

According to the fundamental theorem of existence and uniqueness of the solution to the system of differential equations (1) with the initial value $(S(0), E(0), I(0), R(0)) = (S_0, E_0, I_0, R_0)$, System (1) has a unique solution due to the continuity of the interaction functions on the right-hand side and their partial derivatives.

Theorem 1: System (1) has uniformly bounded solutions.

Proof: Consider the solution of system (1) that is given by $(S(t), E(t), I(t), R(t))$, and let $N(t) = S(t) + E(t) + I(t) + R(t)$, then by the derivative $N(t)$ for time t, it is obtained:

$$\frac{dN}{dt} = \frac{ds}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}.$$

After compensation, it has arrived at:

$$\frac{dN}{dt} = (1-\theta)\Omega + \theta\Omega - \mu[S + E + I + R] - \gamma I - \psi R.$$

Therefore,

$$\frac{dN}{dt} = \Omega - \mu N - \gamma I - \psi R \leq \Omega - \mu N.$$

That can be written as $\frac{dN}{dt} + \mu N \leq \Omega$.

Therefore, using the lemma (2.1) [29] yields that $N(t) \leq \frac{\Omega}{\mu} + Ce^{-\mu t}$, where $C = \frac{\Omega}{\mu}\left(\frac{\mu}{\Omega}N(0) - 1\right)$.

Thus $N \leq \frac{\Omega}{\mu}$, as $t \to \infty$. So, the system (1) solutions have a uniformly bounded solution.

Theorem 2: For any $t \geq 0$, all systems' (1) solutions with positive initial conditions are positive.

Proof: The general solution of the first equation of the system (1) using the initial value $S_0 > 0$, $E_0 > 0$, $I_0 > 0$, and $R_0 > 0$ can be determined as:

$$\frac{dS}{dt} = (1-\theta)\Omega - \frac{\beta SI}{1+bI} - \mu S + \psi R \geq -S\left(\frac{\beta I}{1+bI} + \mu\right)$$

Then:

$$S(t) \geq S_0 e^{\int_0^t -\left(\frac{\beta I(p)}{1+bI(p)} + \mu\right) dp} > 0.$$

Similarly, for the other functions of system (1), it is obtained that:

$$E(t) \geq E_0 e^{\int_0^t -(\mu+\delta)\, dp} > 0.$$

$$I(t) \geq I_0 e^{\int_0^t -\left(\frac{\phi u}{1+\alpha u} + \mu + \rho + \gamma\right) dp} > 0.$$

$$R(t) \geq R_0 e^{\int_0^t -(\mu+\psi)\, dp} > 0.$$

Hence, it is obtained that for all positive initial values, the solutions still positive.

3. Equilibria and Basic Reproduction Number

From the system (1), it is easy to verify that there are two equilibrium points: the disease-free equilibrium point defined by $\rho_0 = (\hat{S},\ 0, 0, \hat{R})$, where:

$$\hat{S} = \frac{\Omega(\mu(1-\theta)+\psi)}{\mu(\mu+\psi)}, \hat{R} = \frac{\theta\Omega}{(\mu+\psi)}. \tag{2}$$

As for the point where the endemic $\rho_1 = (S^*, E^*, I^*, R^*)$ in the system (1), it is determined by:

$$\left.\begin{aligned} S^* &= \frac{(1+bI^*)[(1+\alpha uI^*)[\mu\Omega(1-\theta)+\psi\Omega+\rho\psi I^*]+u\phi\psi I^*]}{[\beta I^*+\mu(1+bI^*)](1+\alpha uI^*)(\mu+\psi)} \\ E^* &= \frac{\beta I^*[(1+\alpha uI^*)[\mu\Omega(1-\theta)+\psi\Omega+\rho\psi I^*]+u\phi\psi I^*]}{[\beta I^*+\mu(1+bI^*)](1+\alpha uI^*)(\mu+\psi)(\mu+\delta)} \\ R^* &= \frac{\theta\Omega(1+\alpha uI^*)+[\phi u+\rho(1+\alpha uI^*)]I^*}{(\mu+\psi)(1+\alpha uI^*)} \end{aligned}\right\}, \tag{3}$$

while I^* is a positive root of a polynomial equation of the third degree:

$$C_1 J^2 + C_2 J + C_2 = 0, \tag{4}$$

where:

$$C_1 = u\alpha\big((\delta+\mu)[\beta\gamma + \delta\mu(\beta + b\gamma) + b\delta\mu^2 + b\mu\rho] + \beta\delta\rho(1-\psi) + \beta\mu\rho\big),$$

$$C_2 = (\delta+\mu)[\beta\gamma + u\alpha\gamma\mu + \mu(\beta + b\gamma) + (b + u\alpha)\mu(\mu+\rho) + \beta(\rho + u\phi) + bu\mu\phi] \\ -\beta\delta\psi(\rho + u\phi) - u\alpha\beta\delta\Omega\big((1-\theta)\mu + \psi\big),$$

$$C_3 = \mu(\delta+\mu)(\gamma+\mu+\rho+u\phi) - \beta\delta\Omega\big((1-\theta)\mu+\psi\big) \\ = \mu(\delta+\mu)(\gamma+\mu+\rho+u\phi)\left[1 - \frac{\beta\delta\Omega\big((1-\theta)\mu+\psi\big)}{\mu(\delta+\mu)(\gamma+\mu+\rho+u\phi)}\right].$$

Consequently, the endemic equilibrium point exists uniquely if the coefficient of equation (4) satisfies the following condition:

$$1 < \frac{\beta\delta\Omega\big((1-\theta)\mu+\psi\big)}{\mu(\delta+\mu)(\gamma+\mu+\rho+u\phi)} = R_1. \tag{5}$$

A key idea in epidemiology, the basic reproduction number measures the potential for an infectious disease to spread. In an entirely susceptible population, it indicates the typical number of secondary infections brought on by a single infected person. To determine the basic reproduction number, denoted (R_0), the next-generation matrix method [30] is used. Write the model (1) in the form:

$$\frac{dZ}{dt} = \mathcal{F}(Z) - \mho(Z),$$

where $Z = (E, I, S, R)^T$ and:

$$\mathcal{F} = \begin{bmatrix} \frac{\beta SI}{1+bI} \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ and } \mho = \begin{bmatrix} \mu E + \delta E \\ -\delta E + \frac{\phi uI}{1+\alpha uI} + \mu I + \rho I + \gamma I \\ -(1-\theta)\Omega + \frac{\beta SI}{1+bI} + \mu S - \psi R \\ -\theta\Omega - \frac{\phi uI}{1+\alpha uI} - \rho I + \mu R + \psi R \end{bmatrix}.$$

Since the disease-free equilibrium $\rho_0 = (\hat{S}, 0, 0, \hat{R})$, then the Jacobian matrix of $\mathcal{F}$ and $\mho$ evaluated at ρ_0 are respectively given by:

$$D\mathcal{F}(Z, \rho_0) = \begin{pmatrix} F & \mathbf{0} \\ \mathbf{0} & \mathbf{0} \end{pmatrix}, \text{ and } D\mho(Z, \rho_0) = \begin{pmatrix} V & \mathbf{0} \\ g_1 & g_2 \end{pmatrix},$$

where $\mathbf{0}$ is a 2×2 zero matrix, $F = \begin{pmatrix} 0 & \beta\hat{S} \\ 0 & 0 \end{pmatrix}$, $V = \begin{pmatrix} \mu+\delta & 0 \\ -\delta & \phi u + \mu + \rho + \gamma \end{pmatrix}$, $g_1 = \begin{pmatrix} 0 & \beta\hat{S} \\ 0 & -\phi u - \rho \end{pmatrix}$, and $g_2 = \begin{pmatrix} \mu & -\psi \\ 0 & \mu + \psi \end{pmatrix}$. Therefore, the following is obtained:

$$FV^{-1} = \frac{1}{(\mu+\delta)(\mu+\rho+\gamma+\phi u)} \begin{pmatrix} \frac{\delta\beta\Omega(\mu(1-\theta)+\psi)}{\mu(\mu+\psi)} & \frac{(\mu+\delta)\beta\Omega(\mu(1-\theta)+\psi)}{\mu(\mu+\psi)} \\ 0 & 0 \end{pmatrix}.$$

Hence, the basic reproduction number is the maximum eigenvalue of FV^{-1}, which is calculated by:

$$R_0 = \frac{\delta\beta\Omega(\mu(1-\theta)+\psi)}{\mu(\mu+\psi)(\mu+\delta)(\mu+\rho+\gamma+\phi u)}, \tag{6}$$

Note that, when $R_0 > 1$, then condition (5) is always satisfied.

4. LOCAL STABILITY

In this section, the local stability of the model (1) is determined around the disease-free equilibrium point and the endemic point with the help of the Jacobian matrix J, which is given by:

$$J = \begin{pmatrix} -\frac{\beta I}{1+bI} - \mu & 0 & -\frac{\beta S}{(1+bI)^2} & \psi \\ \frac{\beta I}{1+bI} & -\delta-\mu & \frac{\beta S}{(1+bI)^2} & 0 \\ 0 & \delta & -\gamma-\mu-\rho-\frac{u\phi}{(1+u\alpha I)^2} & 0 \\ 0 & 0 & \rho+\frac{u\phi}{(1+u\alpha I)^2} & -\mu-\psi \end{pmatrix}, \tag{7}$$

Now, after the Jacobian is determined, the local stability of the disease-free point ρ_0 is considered in the following theorem.

Theorem 3: If the condition $R_0 < 1$ holds, the disease-free point $\rho_0 = (\hat{S}, 0, 0, \hat{R})$ becomes locally asymptotically stable, and it is unstable otherwise.

Proof: The Jacobian matrix (7), at the point ρ_0, becomes:

$$J(\rho_0) = \begin{pmatrix} -\mu & 0 & -\beta\hat{S} & \psi \\ 0 & -\delta-\mu & \beta\hat{S} & 0 \\ 0 & \delta & -\gamma-\mu-\rho-u\phi & 0 \\ 0 & 0 & \rho+u\phi & -\mu-\psi \end{pmatrix} \tag{8}$$

Thus, the characteristic equation can be written:

$$(-\mu-\lambda)(-(\mu+\psi)-\lambda)(\lambda^2 - T\lambda + D) = 0, \tag{9}$$

where $T = -2\mu-\delta-\gamma-\rho-\phi u < 0$, and $D = (\mu+\delta)(\phi u+\mu+\gamma+\rho) - \beta\delta\hat{S}$. Then, by substituting the value of $\hat{S}$, it yields that $D = (\mu+\delta)(\phi u+\mu+\gamma+\rho)[1-R_0]$.

Therefore, all the eigenvalues of the $J(\rho_0)$ are negative when the condition $R_0 < 1$. Thus, the model (1) is locally asymptotically stable under the given condition, and it is unstable otherwise.

Theorem 4: If the following conditions hold, then the endemic point $\rho_1 = (S^*, E^*, I^*, R^*)$ of the model (1) is locally asymptotically stable.

$$\frac{\beta S^*}{(1+bI^*)^2} + \psi < \frac{\beta I^*}{1+bI^*} + \mu. \tag{10}$$

$$\frac{\beta I^*}{1+bI^*}+\frac{\beta S^*}{(1+bI^*)^2}<\delta+\mu. \tag{11}$$

$$\delta<\gamma+\mu+\rho+\frac{u\phi}{(1+u\alpha I^*)^2}. \tag{12}$$

$$\rho+\frac{u\phi}{(1+u\alpha I^*)^2}<\mu+\psi. \tag{13}$$

Proof: The Jacobian matrix (7), around the point ρ_1, becomes

$$J(\rho_1)=\begin{pmatrix} -\frac{\beta I^*}{1+bI^*}-\mu & 0 & -\frac{\beta S^*}{(1+bI^*)^2} & \psi \\ \frac{\beta I^*}{1+bI^*} & -\delta-\mu & \frac{\beta S^*}{(1+bI^*)^2} & 0 \\ 0 & \delta & -\gamma-\mu-\rho-\frac{u\phi}{(1+u\alpha I^*)^2} & 0 \\ 0 & 0 & \rho+\frac{u\phi}{(1+u\alpha I^*)^2} & -\mu-\psi \end{pmatrix}=(a_{ij}). \tag{14}$$

Now, according to the Gershgorin disc theorem [31], with radius equal to the total of the absolute values of the non-diagonal entries in the i^{th} row, each eigenvalue of $J(\rho_1)$ falls inside at least one of the Gershgorin discs centered at a_{ii}.

Now, since all the diagonal entries a_{ii} are negative. Then, according to the given conditions, all the Gershgorin discs are located entirely on the left-hand side of the complex plane.

Therefore, each eigenvalue has a negative real part, and hence ρ_1 is locally asymptotically stable.

5. GLOBAL STABILITY

Global stability analysis, as used in epidemiological models, is the mathematical examination of a dynamical system's long-term behavior from any initial condition. Whatever the location where the disease begins, it determines whether the system will converge to a specific steady state, such as one in which the disease is absent from the population or one in which the disease persists functionally, as shown in the following theorems:

Theorem 5: The point V_0 is a globally asymptotically stable equilibrium provided that:

$$R_0<R_2<1, \tag{15}$$

with $R_2=\left(\mu+\gamma+\rho+\frac{\phi u}{1+\alpha u I_{max}}\right)$.

Proof: Consider the positive semi-definite function:

$$L_0(S,E,I,R)=C_1E+C_2I\ .$$

Note that $L_0: R_+^4\to R$ with $L_0(\rho_0)=0$, and $L_0(S,E,I,R)\geq 0$ otherwise. Additionally, the derivative of L_0 for its input:

$$\frac{dL_0}{dt} = \frac{dE}{dt} + \frac{dI}{dt}.$$

Moreover, by using the proposed algorithm in [35], it is found that:

$$\begin{aligned}\frac{dL_0}{dt} &= \left(\frac{\beta SI}{1+bI} - \mu E - \delta E\right) + \left(\delta E - \frac{\phi uI}{1+\alpha uI} - \mu I - \gamma I - \rho I\right) \\ &\leq -\mu E + \beta \hat{S} I - \left(\frac{\phi u}{1+\alpha u I_{max}} + \mu + \gamma + \rho\right) I,\end{aligned}$$

where I_{max} is the upper bound of the population I resulting from Theorem 1. Now, it is obtained that:

$$\frac{dL_0}{dt} < -\mu E - \left(\mu + \gamma + \rho + \frac{\phi u}{1+\alpha u I_{max}}\right)\left(1 - \frac{\beta \hat{S}}{\left(\mu+\gamma+\rho+\frac{\phi u}{1+\alpha u I_{max}}\right)}\right) I.$$

Therefore, due to the given condition, $\frac{dL_0}{dt}$ will be a negative semidefinite function. When the specified condition is met, ρ_0 is globally asymptotically stable according to LaSalle's invariance principle [32].

Now, in the following theorem, the global stability of ρ_1 is discussed.

Theorem 6: The endemic equilibrium point ρ_1 has a basin of attraction that satisfies the following requirements:

$$\left[\frac{\beta S}{EAA^*} - \frac{\delta}{I}\right]^2 < \frac{4}{6}\left[\frac{\mu}{E} + \frac{\delta}{E}\right]\left[\frac{\mu}{I} + \frac{\gamma}{I} + \frac{\rho}{I} + \frac{\phi u}{BB^*I}\right]. \tag{16}$$

$$\left(\frac{\psi}{S}\right)^2 < \frac{4}{6}\left[\frac{\mu}{S} + \frac{\beta I^*}{SA^*}\right]\left[\frac{\mu}{R} + \frac{\psi}{R}\right]. \tag{17}$$

$$\left[\frac{\phi u}{BB^*R} - \frac{\rho}{R}\right]^2 < \frac{4}{6}\left[\frac{\mu}{I} + \frac{\gamma}{I} + \frac{\rho}{I} + \frac{\phi u}{BB^*I}\right]\left[\frac{\mu}{R} + \frac{\psi}{R}\right]. \tag{18}$$

$$\left(\frac{\beta}{AA^*}\right)^2 < \frac{4}{9}\left[\frac{\mu}{S} + \frac{\beta I^*}{SA^*}\right]\left[\frac{\mu}{I} + \frac{\gamma}{I} + \frac{\rho}{I} + \frac{\phi u}{BB^*I}\right]. \tag{19}$$

$$\left(\frac{\beta I^*}{EA^*}\right)^2 < \frac{4}{6}\left[\frac{\mu}{S} + \frac{\beta I^*}{SA^*}\right]\left[\frac{\mu}{E} + \frac{\delta}{E}\right]. \tag{20}$$

Proof: Consider the positive definite function:

$$\begin{aligned}L_1 = &\left(S - S^* - S^* ln\frac{S}{S^*}\right) + \left(E - E^* - E^* ln\frac{E}{E^*}\right) \\ &+ \left(I - I^* - I^* ln\frac{I}{I^*}\right) + \left(R - R^* - R^* ln\frac{R}{R^*}\right).\end{aligned}$$

Note that $L_1 : R_+^4 \to R$ with $L_1(\rho_1) = 0$, and $L_1(x) > 0$ otherwise. Additionally, the derivative of L_1 for its input:

$$\frac{dL_1}{dt} = \frac{\partial L_1}{\partial S}\frac{dS}{dt} + \frac{\partial L_1}{\partial E}\frac{dE}{dt} + \frac{\partial L_1}{\partial I}\frac{dI}{dt} + \frac{\partial L_1}{\partial R}\frac{dR}{dt}.$$

Hence, after substituting the system equations, it is obtained that:

$$\frac{dL_1}{dt} = -\left[\frac{\mu}{S} + \frac{\beta I^*}{SA^*}\right](S-S^*)^2 - \left[\frac{\mu}{E} + \frac{\delta}{E}\right](E-E^*)^2 - \left[\frac{\mu}{I} + \frac{\gamma}{I} + \frac{\rho}{I} + \frac{\phi u}{BB^*I}\right](I-I^*)^2$$
$$-\left[\frac{\mu}{R} + \frac{\psi}{R}\right](R-R^*)^2 - \left[\frac{\beta S}{EAA^*} - \frac{\delta}{I}\right](E-E^*)(I-I^*)$$
$$-\left[\frac{\phi u}{BB^*R} - \frac{\rho}{R}\right](I-I^*)(R-R^*)$$
$$+\left[\frac{\psi}{S}(R-R^*) - \frac{\beta}{AA^*}(I-I^*) + \frac{\beta I^*}{EA^*}(E-E^*)\right](S-S^*),$$

where $A = 1 + bI$, $A^* = 1 + bI^*$, $B = 1 + \alpha uI$, and $B^* = 1 + \alpha uI^*$.

$$\frac{dL_1}{dt} \leq -\left[\frac{\mu}{S} + \frac{\beta I^*}{SA^*}\right](S-S^*)^2 - \left[\frac{\mu}{E} + \frac{\delta}{E}\right](E-E^*)^2 - \left[\frac{\mu}{I} + \frac{\gamma}{I} + \frac{\rho}{I} + \frac{\phi u}{BB^*I}\right](I-I^*)^2$$
$$-\left[\frac{\mu}{R} + \frac{\psi}{R}\right](R-R^*)^2 - \left[\frac{\beta S}{EAA^*} - \frac{\delta}{I}\right](E-E^*)(I-I^*)$$
$$+\frac{\psi}{S}(S-S^*)(R-R^*) - \left[\frac{\phi u}{BB^*R} - \frac{\rho}{R}\right](I-I^*)(R-R^*)$$
$$-\frac{\beta}{AA^*}(S-S^*)(I-I^*) + \frac{\beta I^*}{EA^*}(S-S^*)(E-E^*).$$

Accordingly, by using the definition of the negative definite quadratic function, the conditions (16)-(20) guarantee that the function $\frac{dL_1}{dt}$ is negative definite. Hence, the proof is done.

6. THE LOCAL BIFURCATION

When a system's parameter (or parameters) gradually moves through significant values, a qualitative shift in the system's dynamical behavior, such as the emergence or disappearance of equilibria or changes in their stability, is referred to as a local bifurcation. Analyzing how disease dynamics alter with changes in important parameters like transmission rates, recovery rates, or population numbers requires an understanding of local bifurcations in the context of epidemiological models. Therefore, in this section, the local bifurcation requirements for the system (1) will be determined. The system is rewritten as follows:

$$\frac{dZ}{dt} = G(Z),\ Z = (z_1, z_2, z_3, z_4)^T = (S, E, I, R)^T, \text{ and } G = (g_1, g_2, g_3, g_4)^T,$$

where g_1, g_2, g_3, g_4 are the functions on the right side of the model (1). Based on that, the second derivative of G for Z can be written:

$$D^2G(Z)(V,V) = \begin{bmatrix} -\frac{2\beta v_1 v_3}{(1+bI)^2} + \frac{2\beta bSv_3^2}{(1+bI)^3} \\ \frac{2\beta v_1 v_3}{(1+bI)^2} - \frac{2\beta bSv_3^2}{(1+bI)^3} \\ \frac{2u^2\alpha\phi v_3^2}{(1+u\alpha I)^3} \\ -\frac{2u^2\alpha\phi v_3^2}{(1+u\alpha I)^3} \end{bmatrix}, \tag{21}$$

where $V = (v_1, v_2, v_3, v_4)^T$ be any non-zero vector.

Theorem 7: System (1) experiences a transcritical bifurcation at the disease-free equilibrium

point ρ_0 when $R_0 = 1$, provided that:

$$2u^2\alpha\phi + 2\frac{(\mu+\rho+\gamma+\phi u)}{\delta\Omega(\mu(1-\theta)+\psi)}(\delta\psi(\rho+u\phi) - (\mu+\delta)(\mu+\psi)(\mu+\rho+\gamma+\phi u) - \delta b\Omega(\mu(1-\theta)+\psi)) \neq 0. \quad (22)$$

In addition, the direction of the transcritical bifurcation is backward provided that:

$$\frac{\psi(\rho+u\phi)}{\mu+\psi} < \frac{2\mu+\delta}{\mu+\delta}\check{\beta}\hat{S}, \quad (23)$$

where $\check{\beta}$ are given in the proof.

Proof: According to $J(\rho_0)$ that is given in Eq. (8) with $R_0 = 1$, then the characteristic equation becomes:

$$(-\mu-\lambda)(-(\mu+\psi)-\lambda)\lambda(\lambda-T) = 0,$$

where $T = -2\mu - \delta - \gamma - \rho - \phi u < 0$, while D, that is given in Eq. (9), becomes:

$$D = (\mu+\delta)(\phi u + \mu + \gamma + \rho)[1 - R_0] = 0.$$

Hence, it has the eigenvalues $\lambda_{01} = -\mu$, $\lambda_{02} = -(\mu+\psi)$, $\lambda_{03} = T$, and $\lambda_{04} = 0$, which means ρ_0 becomes a non-hyperbolic point.

Let $V_{01} = (v_1, v_2, v_3, v_4)^T$, and $\Psi_{01} = (\psi_1, \psi_2, \psi_3, \psi_4)^T$, be the eigenvectors corresponding to $\lambda_{04} = 0$ for $[J(\rho_0)]$, and $[J(\rho_0)]^T$ respectively. Direct computation yields that:

$$V_{01} = \begin{pmatrix} \frac{\psi}{\mu}\frac{\rho+u\phi}{\mu+\psi} - \frac{\beta\hat{S}}{\mu} \\ \frac{\beta\hat{S}}{\mu+\delta} \\ 1 \\ \frac{\rho+u\phi}{\mu+\psi} \end{pmatrix}, \ \Psi_{01} = \begin{pmatrix} 0 \\ \frac{\delta}{\mu+\delta} \\ 1 \\ 0 \end{pmatrix}.$$

Moreover, from $R_0 = 1$, the parameter $\check{\beta} = \frac{\mu(\mu+\psi)(\mu+\delta)(\mu+\rho+\gamma+\phi u)}{\delta\Omega(\mu(1-\theta)+\psi)}$ is selected as a bifurcation parameter, then the following results are obtained:

$$\frac{dG}{d\beta} = G_\beta = \begin{bmatrix} \frac{-SI}{1+bI} \\ \frac{SI}{1+bI} \\ 0 \\ 0 \end{bmatrix} \Rrightarrow G_\beta(\rho_0, \check{\beta}) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

$$DG_\beta(\rho_0, \check{\beta}) = \begin{bmatrix} 0 & 0 & -\hat{S} & 0 \\ 0 & 0 & \hat{S} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Then, by using Eq. (21), it is obtained that:

$$D^2G(\rho_0,\check{\beta})(V_{01},V_{01})=\begin{bmatrix}-2\check{\beta}\left(\frac{\psi(\rho+u\phi)-\check{\beta}(\mu+\psi)\hat{S}}{\mu(\mu+\psi)}\right)+2\check{\beta}b\hat{S}\\ 2\check{\beta}\left(\frac{\psi(\rho+u\phi)-\check{\beta}(\mu+\psi)\hat{S}}{\mu(\mu+\psi)}\right)-2\check{\beta}b\hat{S}\\ 2u^2\alpha\phi\\ -2u^2\alpha\phi\end{bmatrix}.$$

Therefore, it is obtained that:

$$\Psi_{01}^T DG_\beta(\rho_0,\check{\beta})=0.$$

$$\Psi_{01}^T DG_\beta(\rho_0,\check{\beta})V_{01}=\frac{\delta\hat{S}}{\mu+\delta}\neq 0.$$

$$\Psi_{01}^T[D^2G(\rho_0)(V_{01},V_{01})]=2u^2\alpha\phi+2\frac{(\mu+\rho+\gamma+\phi u)}{\delta\Omega(\mu(1-\theta)+\psi)}(\delta\psi(\rho+u\phi)$$
$$-(\mu+\delta)(\mu+\psi)(\mu+\rho+\gamma+\phi u)-\delta b\Omega(\mu(1-\theta)+\psi)).$$

According to the Sotomayor Theorem [33], it has a transcortical bifurcation under the condition (22).

Now, Theorem 4.1 in [34] is applied to describe the direction of the bifurcation at $R_0=1$ or $\beta=\check{\beta}$ near the point ρ_0.

Because the other eigenvalue is real and negative, while $\lambda_{04}=0$ is a simple zero eigenvalue. The disease-free equilibrium ρ_0 is therefore a non-hyperbolic equilibrium when $R_0=1$ or $\beta=\check{\beta}$, confirming the first assumption of Theorem 4.1 in [34].

Since V_{01} determines the right eigenvector linked to $\lambda_{04}=0$, hence $U_{01}=(u_1,u_2,u_3,u_4)$, the left eigenvector linked to $\lambda_{04}=0$ that satisfies $U_{01}V_{01}=1$ can be determined as $U_{01}=(0,0,1,0)$.

Moreover, the coefficients $\hat{a}$ and $\hat{b}$, which specify the direction of the bifurcation, are given in [34]:

$$\left.\begin{aligned}\hat{a}&=\sum_{k,i,j=1}^{n}u_k v_i v_j\frac{\partial^2 g_k}{\partial z_i\partial z_j}(\rho_0,\check{\beta})\\ \hat{b}&=\sum_{i,j=1}^{n}v_i v_j\frac{\partial^2 g_i}{\partial z_j\partial\beta}(\rho_0,\check{\beta})\end{aligned}\right\}. \tag{24}$$

Direct computation shows that for the system (1):

$$\hat{a}=2u^2\alpha\phi>0. \tag{25}$$

$$\hat{b}=\left[-\frac{\psi(\rho+u\phi)}{\mu+\psi}+\frac{2\mu+\delta}{\mu+\delta}\check{\beta}\hat{S}\right]\frac{\hat{S}}{\mu}. \tag{26}$$

Since $\hat{a}$ is always positive, the sign of the coefficient $\hat{b}$ determines the local dynamics surrounding ρ_0 for $R_0=1$. Consequently, a backward bifurcation at $R_0=1$ is certain to occur under the condition (23) and the prof is complete.

Theorem 8: System (1) experiences a saddle–node bifurcation near the endemic equilibrium point ρ_1 when ψ passes a positive value $\frac{-\mu[a_{11}a_{22}a_{33}+a_{13}a_{21}a_{32}-a_{11}a_{23}a_{32}]}{a_{21}a_{32}a_{43}+a_{11}a_{22}a_{33}+a_{13}a_{21}a_{32}-a_{11}a_{23}a_{32}} \equiv (\psi^*)$ provided that:

$$a_{22}a_{33} - a_{23}a_{32} > 0. \tag{27}$$

$$-(\xi_4 - \xi_5)\left[\frac{2\beta\xi_3(\xi_1 - bS^*\xi_3)}{(1+bI^*)^2}\right] + \frac{2u^2\alpha\phi\xi_3^2}{(1+u\alpha I^*)^3}(\xi_6 - 1) \neq 0. \tag{28}$$

Proof: Consider the Jacobian matrix of System (1) for the endemic equilibrium points ρ_1, that is given by Eq. (14), it has the following characteristic polynomial:

$$\lambda^4 + c_1\lambda^3 + c_2\lambda^2 + c_3\lambda + c_4 = 0,$$

where:

$$c_1 = -[a_{11} + a_{22} + a_{33} + a_{44}^*],$$

$$c_2 = a_{11}(a_{22} + a_{33} + a_{44}^*) + a_{22}a_{44}^* + a_{33}a_{44}^* + a_{22}a_{33} - a_{32}a_{23},$$

$$c_3 = -[a_{11}a_{22}a_{33} + a_{13}a_{21}a_{32} + a_{22}a_{33}a_{44}^* - a_{11}a_{23}a_{32} - a_{23}a_{32}a_{44}^* + a_{11}a_{33}a_{44}^* - a_{11}a_{34}a_{43} + a_{11}a_{22}a_{44}^*],$$

$$c_4 = (a_{11}a_{22}a_{33} - a_{11}a_{23}a_{32} + a_{13}a_{21}a_{32})a_{44}^* - a_{14}^*a_{21}a_{32}a_{43}.$$

Here a_{ij} are the coefficients of the $J(\rho_1, \psi^*) = J_1$ with $a_{44}^* = a_{44}(\psi^*)$, and $a_{14}^* = a_{14}(\psi^*)$. It is easy to verify that J_1 has a zero eigenvalue $\lambda^* = 0$ when $\psi = \psi^*$, which leads $c_4 = 0$. Hence, ρ_1 becomes a non-hyperbolic point, and the necessary but not sufficient condition of the bifurcation is obtained.

Let $V_{11} = (v_{11}, v_{12}, v_{13}, v_{14})^T$, and $\Psi_{11} = (\psi_{11}, \psi_{12}, \psi_{13}, \psi_{14})^T$, be the eigenvectors corresponding to $\lambda^* = 0$ for J_1, and $[J_1]^T$ respectively. By using condition (27) and the signs of the coefficients, direct computation yields that:

$$V_{11} = \begin{pmatrix} \xi_1 \\ \xi_2 \\ \xi_3 \\ 1 \end{pmatrix}, \ \Psi_{11} = \begin{pmatrix} \xi_4 \\ \xi_5 \\ \xi_6 \\ 1 \end{pmatrix},$$

where, $\xi_1 = \frac{a_{14}^*(a_{22}a_{33} - a_{23}a_{32})}{-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32}} > 0$, $\xi_2 = \frac{-a_{21}a_{14}^*a_{33}}{-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32}} > 0$,

$\xi_3 = \frac{a_{14}^*a_{21}a_{32}}{-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32}} > 0$, $\xi_4 = \frac{a_{21}a_{32}a_{43}}{a_{11}(-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32})} < 0$,

$\xi_5 = \frac{-a_{32}a_{43}}{a_{22}(-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32})} > 0$, $\xi_6 = \frac{a_{43}}{-a_{11}a_{22}a_{33}+a_{11}a_{23}a_{32}-a_{13}a_{21}a_{32}} > 0.$

Moreover, with the help of Eq. (21), it is obtained:

$$\Psi_{11}^T G_\psi(\rho_1, \psi^*) = R^*(\xi_4 - 1) < 0,$$

$$\Psi_{11}^{T}[D^2G_{\psi}(\rho_1,\psi^*)(U_{11},U_{11})] = -(\xi_4-\xi_5)\left[\frac{2\beta\xi_3(\xi_1-bS^*\xi_3)}{(1+bI^*)^2}\right]+\frac{2u^2\alpha\phi\xi_3^2}{(1+u\alpha I^*)^3}(\xi_6-1).$$

According to condition (28), all the necessary and sufficient conditions of the Saddle-node bifurcation are satisfied in the sense of Sotomayor's Theorem, which completes the proof.

7. SENSITIVITY ANALYSIS

A key element in the creation and use of epidemiological models is sensitivity analysis. During infections, it helps evidence-based community health decision-making, improves comprehension of model behavior, and boosts certainty in predictions. It is used to determine the important parameters that have the most effect. A local sensitivity analysis is based on the standardized forward sensitivity index, R_0. The sensitivity index of R_0 for the model's parameters is obtained using the following formulas [14].

$$\Lambda_{\omega}^{R_0} = \left(\frac{\partial R_0}{\partial \omega}\right)\left(\frac{\omega}{R_0}\right), \tag{29}$$

where ω is a system parameter divided by R_0. It also $\Lambda_{\omega}^{R_0}$ represents the degree of susceptibility to change in ω. An increase in the parameter value leads to either an increase or a decrease in the value of R_0. Now, can determine $\Lambda_{\omega}^{R_0}$ where we obtain:

$$\Lambda_{\theta}^{R_0} = -\frac{\theta\mu}{(\mu(1-\theta)+\psi)}.$$

$$\Lambda_{\Omega}^{R_0} = 1.$$

$$\Lambda_{\beta}^{R_0} = 1.$$

$$\Lambda_{b}^{R_0} = 0.$$

$$\Lambda_{\mu}^{R_0} = -\frac{\theta\psi\mu}{(\mu+\psi)(\mu(1-\theta)+\psi)}-\frac{\mu(\delta+\mu)+(\delta+2\mu)(\gamma+\mu+\rho+u\phi)}{(\delta+\mu)(\gamma+\mu+\rho+u\phi)}.$$

$$\Lambda_{\psi}^{R_0} = \frac{\theta\mu\psi}{(\mu+\psi)(\mu(1-\theta)+\psi)}.$$

$$\Lambda_{\delta}^{R_0} = \frac{\mu}{(\delta+\mu)}.$$

$$\Lambda_{u}^{R_0} = -\frac{u\phi}{(\gamma+\mu+\rho+u\phi)}.$$

$$\Lambda_{\alpha}^{R_0} = 0.$$

$$\Lambda_{\phi}^{R_0} = -\frac{\phi u}{(\gamma+\mu+\rho+u\phi)}.$$

$$\Lambda_{\rho}^{R_0} = -\frac{\rho}{(\gamma+\mu+\rho+u\phi)}.$$

$$\Lambda_{\gamma}^{R_0} = -\frac{\gamma}{(\gamma+\mu+\rho+u\phi)}.$$

Consequently, we examined the basic reproduction number of System (1). Hence, according to the set of hypothetical parameter values used in the numerical simulation section, the following results of sensitivity regarding their parameters are obtained:

$$\Lambda_{\theta}^{R_0} = -0.14285714285714285.$$

$$\Lambda_{\Omega}^{R_0} = 1.$$

$$\Lambda_{\beta}^{R_0} = 1.$$

$$\Lambda_{b}^{R_0} = 0.$$

$$\Lambda_{\mu}^{R_0} = -1.7806484295845995.$$

$$\Lambda_{\psi}^{R_0} = 0.07142857142857144.$$

$$\Lambda_{\delta}^{R_0} = 0.666666666666667.$$

$$\Lambda_{u}^{R_0} = -0.6382978723404255.$$

$$\Lambda_{\alpha}^{R_0} = 0.$$

$$\Lambda_{\phi}^{R_0} = -0.6382978723404253.$$

$$\Lambda_{\rho}^{R_0} = -0.10638297872340423.$$

$$\Lambda_{\gamma}^{R_0} = -0.2127659574468085.$$

Consequently, the sensitivity indicators in Figure 1 are constructed using the parameter values given by Equation (30). However, as some of the factors change, so may the sensitivity indicators. According to Figure 1, the factors that are positively proportional to the illness outbreak $(R_0 > 1)$ are β, Ω, ψ, and δ. Conversely, the collection of elements that are inversely proportional to the disease outbreak is provided by θ, μ, u, ϕ, ρ, and γ. Finally, the remaining parameters do not affect the disease outbreak.

$$\begin{aligned}&\theta = 0.25, \Omega = 25, \beta = 0.03, b = 1, \mu = 0.1, \psi = 0.1,\\ &\delta = 0.05, \phi = 0.75, u = 2, \alpha = 1, \rho = 0.25, \gamma = 0.5.\end{aligned} \tag{30}$$

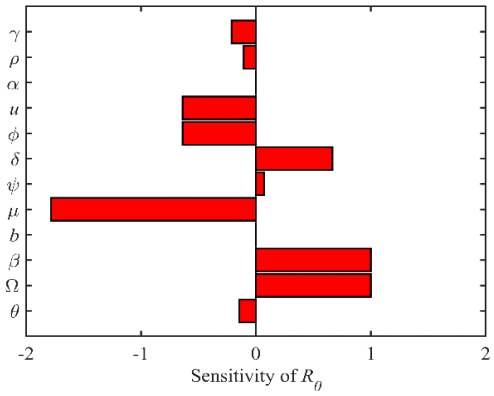

Fig. 1. Sensitivity diagram of R_0 for all system parameters.

According to Figure 1, increasing the parameters β, Ω, ψ, and δ leads to the outbreak of the disease infection due to raising the value of R_0 to greater than or equal to unity.

8. NUMERICAL SIMULATION

When examining epidemiological models, numerical simulation is essential because it enables researchers to assess, forecast, and comprehend the dynamics of disease transmission within populations. Predicting disease spread, testing hypothetical scenarios, assessing control measures, understanding complex dynamics, conducting parameter sensitivity analysis, calibrating and validating models with actual data, and supporting public health policy are the primary objectives of utilizing numerical simulations in epidemiology.

Consequently, using the hypothetical set of data (30), the system (1) is solved numerically starting from various initial points, and then the obtained trajectories are drawn as a function of time, see Figure 2.

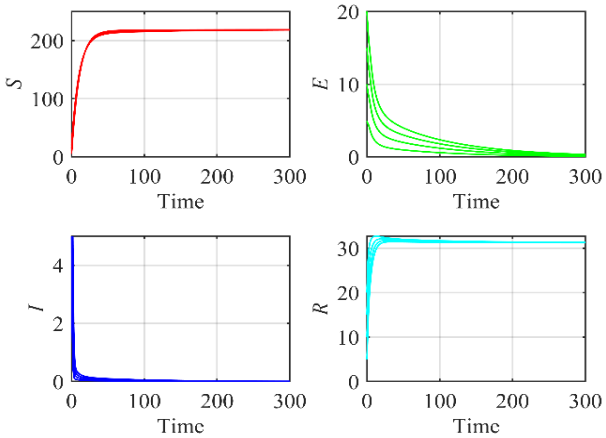

Figure 2: The trajectories of system (1) vs time using data set (30) and starting from different initial points approach $\rho_0 = (218.68, 0,0,31.25)$ with $R_0 = 0.93$.

It is observed through Figure 2 that system 1 approaches asymptotically the disease-free point from different initial points because the $R_0 < 1$. Now, to verify the objectives of using numerical simulation, the parameter values given by Eq. (20) will be changed one at a time, and system (1) will be solved numerically, and then he obtained trajectories will be drawn. It is observed that for $\theta \leq 0.12$, the system approaches ρ_1 as the value of $R_0 \geq 1$, while it approaches ρ_0 otherwise due to the value of $R_0 < 1$, see Figure 3.

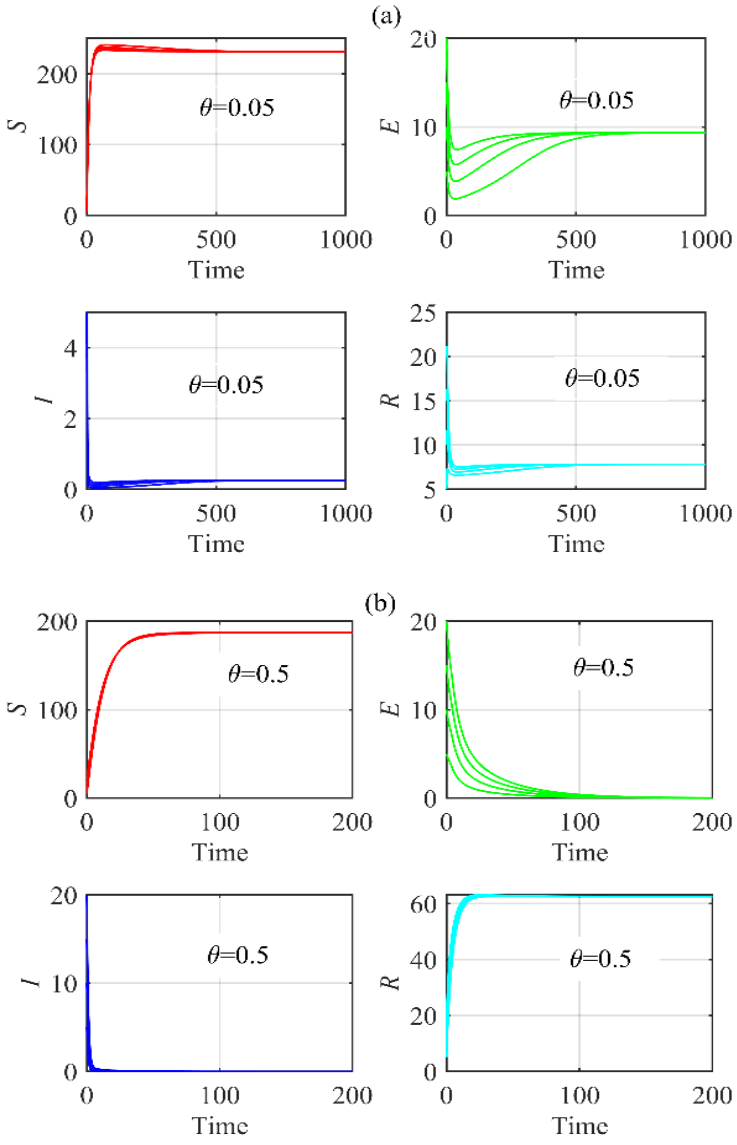

Figure 3. The trajectories of system (1) vs time using data set (30) with different values of θ, and starting from different initial points, (a) They approach $\rho_1 = (231.3, 9.34, 0.25, 7.82)$ with $R_0 = 1.03$, when $\theta = 0.05$. (b) They approach $\rho_0 = (187.46, 0, 0, 62.5)$ with $R_0 = 0.79$, when $\theta = 0.5$.

Note that Figure 3 confirms the results of the sensitivity analysis, as the increase of parameter θ negatively affects the outbreak of disease. Similarly, impact is obtained for the parameter values μ, u, ϕ, ρ, and γ.

Moreover, it is observed that for $\beta \leq 0.032$, the system approaches ρ_0 as the value of $R_0 < 1$, while it approaches ρ_1 otherwise, due to the value of $R_0 \geq 1$, see Figure 4.

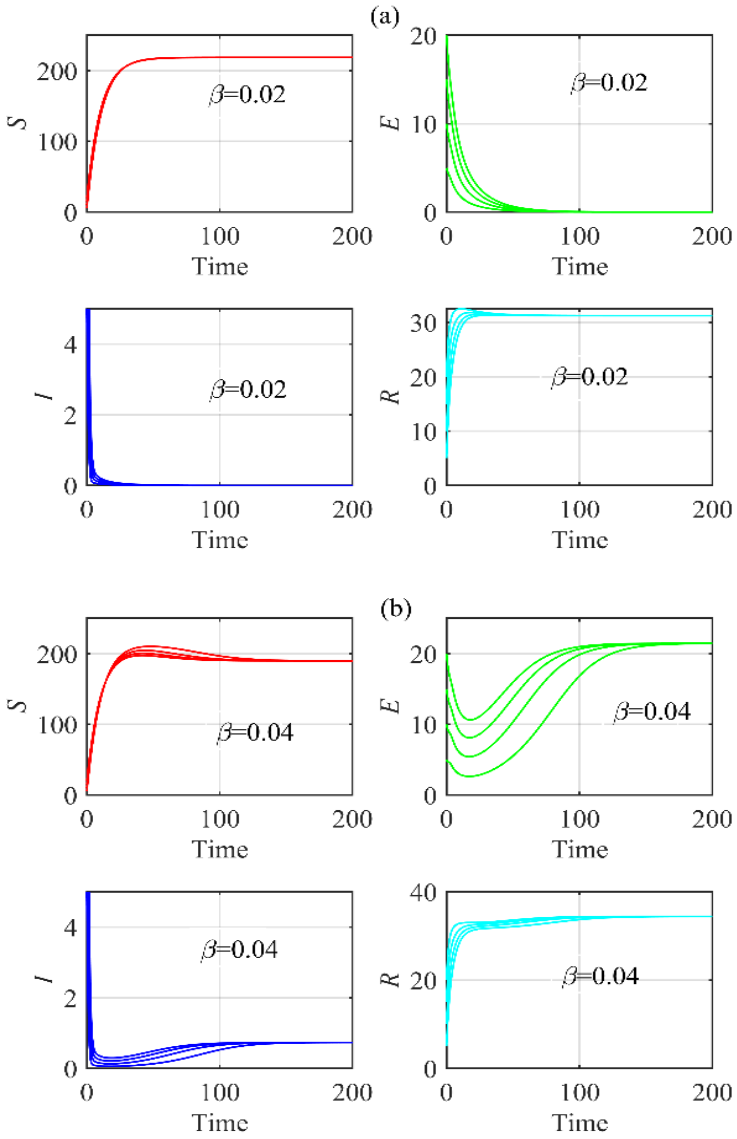

Figure 4. The trajectories of system (1) vs time using data set (30) with different values of β, and starting from different initial points, (a) They approach $\rho_0 = (218.74, 0,0,31.25)$ with $R_0 0.62$, when $\beta = 0.02$. (b) They approach $\rho_1 = (189.71, 21.46,0.73,34.4)$ with $R_0 = 1.24$, when $\beta = 0.04$.

Again, Figure 4 confirms the results of the sensitivity analysis, as the increase of parameter β positively affects the outbreak of disease. Similarly, impact is obtained for the parameter values Ω, ψ, and δ.

Ultimately, although the parameters b and α do not affect the value R_0 as shown in the sensitivity analysis, they do affect the dynamical system through the existence of an endemic point and then backward bifurcation, see Figure 5.

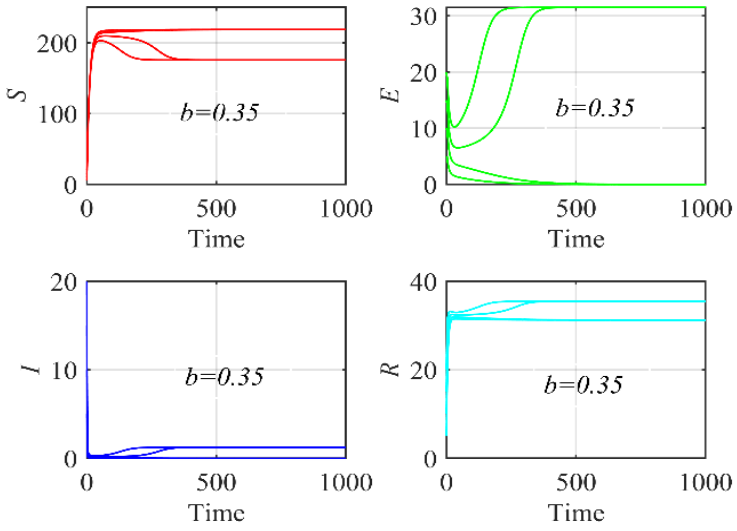

Figure 5. The trajectories of system (1) vs time using data set (30) with $b = 0.35$ and starting from different initial points approach $\rho_0 = (218.74, 0,0,31.25)$ and $\rho_1 = (175.69, 31.49,1.22,35.44)$ with $R_0 = 0.93$.

From Figure 5, it is clear that both equilibria exist even when $R_0 = 0.93 < 1$, and the stability of them depend on the initial points so that the trajectories starting from $(5,5,5,5)$ and $(10,10,10,10)$ approach ρ_0 while those starting from $(15,15,15,15)$ and $(20,20,20,20)$ approach ρ_1, which indicate too bi-stable dynamics and the existence of backward bifurcation. Similarly, results were obtained with varying α, ρ, and γ.

Further analysis has been done, and the obtained results are summarized in Table 2.

Table 2: Dynamical behavior of the system (1) as a function of the parameters changed.

The parameter range	**Dynamical behavior**	**R_0 range**
$\Omega < 26.9$	System (1) approaches ρ_0	$R_0 < 1$
$\Omega \geq 26.9$	System (1) approaches ρ_1	$R_0 \geq 1$
$\mu \leq 0.096$	System (1) approaches ρ_1	$R_0 \geq 1$
$\mu > 0.096$	System (1) approaches ρ_0	$R_0 < 1$

$\psi < 0.32$	System (1) approaches ρ_0	$R_0 < 1$
$\psi \geq 0.32$	System (1) approaches ρ_1	$R_0 \geq 1$
$\delta < 0.056$	System (1) approaches ρ_0	$R_0 < 1$
$\delta \geq 0.056$	System (1) approaches ρ_1	$R_0 \geq 1$
$\phi \leq 0.66$	System (1) approaches ρ_1	$R_0 \geq 1$
$\phi > 0.66$	System (1) approaches ρ_0	$R_0 < 1$
$u \leq 1.78$	System (1) approaches ρ_1	$R_0 \geq 1$
$u > 1.78$	System (1) approaches ρ_0	$R_0 < 1$
$\alpha < 1.7$	System (1) approaches ρ_0	$R_0 < 1$
$\alpha \geq 1.7$	System (1) approaches (ρ_0 & ρ_1)	$R_0 < 1$ (backward)
$\rho \leq 0.087$	System (1) approaches ρ_1	$R_0 \geq 1$
$0.088 \leq \rho \leq 0.094$	System (1) approaches (ρ_0 & ρ_1)	$R_0 < 1$ (backward)
$0.094 < \rho$	System (1) approaches ρ_0	$R_0 < 1$
$\gamma \leq 0.33$	System (1) approaches ρ_1	$R_0 \geq 1$
$\gamma = 0.34$	System (1) approaches (ρ_0 & ρ_1)	$R_0 < 1$ (backward)
$0.34 < \gamma$	System (1) approaches ρ_0	$R_0 < 1$

9. CONCLUSION

In this study, we looked at a saturated incidence SEIR epidemic model that used therapy and vaccination as the two main methods to prevent the spread of infection. The results showed that the model has two equilibria: an endemic equilibrium when the basic reproduction number is exceeded under certain conditions, and a disease-free equilibrium when it is less than one. It is known that the endemic point is locally stable under certain circumstances, whereas the disease-free point is locally asymptotically stable when $R_0 < 1$ and unstable otherwise. The endemic point is demonstrated to have a basin of attraction, whereas the global stability condition for the disease-free point is achieved. Furthermore, mathematical studies showed the possibility of back branching, which complicates illness control initiatives if R_0 decline is the sole criterion taken into consideration without taking other system-affecting elements into account. Sensitivity analysis has revealed the most significant disease dynamics coefficients, providing decision makers with a helpful tool to focus treatments on more sensitive elements. According to numerical simulations, increasing vaccination rates and offering efficient treatment can greatly slow the disease's spread, particularly when done as part of a comprehensive plan. These results suggest that balanced treatment and prevention strategies based on accurate data on the behavior

and responsiveness to changes in the epidemiological system are essential for the effective control of infectious illnesses. When creating reaction plans for impending outbreaks, this model is a useful theoretical tool.

Furthermore, numerical simulation indicates that lower disease spread results from increasing the parameter values of the disease-caused death rate, disease recovery rate, treatment control function, treatment effectiveness, maximum energy therapy function, natural death rate, and the rate of individuals vaccinated at birth. On the other hand, sickness spreads as the values of the parameters (the infected rate, the likelihood of losing immunity, the transmission rate, and the recruitment rate) increase. Additionally, the saturation factor and the saturation parameter in the treatment are backward bifurcation parameters, even though they do not affect the value of R_0. Their appearance at the endemic point is the reason for this.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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