ADV MATH SCI JOURNAL
Advances in Mathematics: Scientific Journal **14** (2025), no.1, 1–18
ISSN: 1857-8365 (printed); 1857-8438 (electronic)
https://doi.org/10.37418/amsj.14.1.1

GLOBAL STABILITY OF AN SEIS EPIDEMIC MODEL WITH BEDDINGTON-DEANGELIS INFECTION RATE

Soufiane Elkhaiar

ABSTRACT. In this paper, the global dynamic behaviors of an SEIS epidemic model with Beddington-DeAngelis infection rate are investigated. This model takes into account constant recruitment, death from the disease, and the phenomenon of latency. Under some hypotheses, it is shown that the global dynamics is determined by the basic reproduction number R_0. If R_0 is less than unity, the disease-free equilibrium is both locally and globally stable and the disease dies out. If R_0 is greater than unity, sufficient conditions for the global stability of the endemic equilibrium are obtained by the geometric approach of Li and Muldowney. Some numerical simulations are also presented to confirm the analytical results.

1. INTRODUCTION

During the last years, mathematical modeling has become a very powerful tool in epidemiology, allowing researchers to predict infectious diseases, assess the impact of interventions, and inform public health policies. By dividing a population into distinct compartments, such as susceptible, infected, vaccinated, quarantined, and recovered individuals, these models provide a quantitative framework for understanding the dynamics of epidemic transmission (see [3] and the references therein).

[1]*corresponding author*
2020 *Mathematics Subject Classification.* 34D20, 34D23.
Key words and phrases. SEIS epidemic model, Beddington-DeAngelis infection rate, global asymptotic stability, Lyapunov functions, geometric approach.
Submitted: 20.12.2024; *Accepted:* 05.01.2025; *Published:* 16.01.2025.

A crucial aspect of many infectious diseases is the existence of an incubation period, during which individuals are infected but not yet infectious. This latency period can influence significantly the spread of the disease. To capture this phenomenon, various modeling approaches have been developed:

(i) Delayed SIR Model: This model incorporates a time delay into the differential equations to represent the incubation period.
(ii) SEIR Model: This model introduces an additional compartment for exposed individuals, who are infected but not yet infectious.

Both approaches provide valuable insights into the dynamics of infectious disease transmission [1, 10].

In the mathematical analysis of epidemiological models, a great interest has been given to the infection rate of susceptible individuals through their contacts with infectious. In many epidemic models, two types are frequently used: the bilinear incidence rate βSI and the standard incidence rate $\dfrac{\beta SI}{N}$. The first one is based on the law of mass action, for the second one, it may be a good approximation if the number of available partners is large enough and everybody could not make more contacts than is practically feasible [22]. It has been suggested by several authors that the disease transmission process may have a saturation incidence rate [4, 21–24].

In 1975, Beddington and DeAngelis ([2, 7]) introduced a the functional $\frac{\beta S(t)I(t)}{1+\alpha_1+\alpha_2 I(t)}$, where β is the transmission rate of disease, α_1 is a measure of inhibition for the susceptible population, and α_2 is a measure of inhibition for the infected population. Beddington-DeAngelis infection rate has been studied by many authors in epidemic models [1, 10, 16, 19], and clearly, this includes three forms:

(i) The bilinear incidence rate βSI where β is the transmission rate ([8, 13]), with $\alpha_1 = \alpha_2 = 0$;
(ii) The saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 S}$ ([23, 24]), with $\alpha_2 = 0$;
(iii) The saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_2 I}$ [4, 11, 21, 22]), with $\alpha_1 = 0$.

In 2001, Fan et al. [8], have studied the SEIS epidemic model with bilinear incidences rates that incorporates constant recruitment, disease-caused death and disease latency:

$$
\begin{cases} \frac{dS}{dt} = \Lambda - \mu S(t) - \beta S(t)I(t) + \delta I(t), \\ \frac{dE}{dt} = \beta S(t)I(t) - (\mu+\sigma)E(t) \\ \frac{dI}{dt} = \sigma E(t) - (\mu+\alpha+\delta)I(t), \end{cases} \tag{1.1}
$$

The global stability of a disease-free equilibrium and an endemic equilibrium was investigated respectively by using Lyapunov function theory and geometric approach. It was proven that the dynamics of 1.1 are completely determined by the basic reproduction number.It was proven that the global dynamics is completely determined by the basic reproduction number R_0 : If $R_0 < 1$, the disease-free equilibrium is globally stable and the disease dies out. If $R_0 > 1$, a unique endemic equilibrium is globally stable in the interior of the feasible region and the disease persists at the endemic equilibrium.

Motivated by the aforementioned considerations, this article explores an SEIS epidemic model incorporating a Beddington-DeAngelis infection rate, as depicted in Figure 1:

$$
\begin{cases} \frac{dS}{dt} = \Lambda - \mu S(t) - \frac{\beta S(t)I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)} + \delta I(t), \\ \frac{dE}{dt} = \frac{\beta S(t)I(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)} - (\mu+\sigma)E(t) \\ \frac{dI}{dt} = \sigma E(t) - (\mu+\alpha+\delta)I(t), \end{cases} \tag{1.2}
$$

where $S(t)$ represents the number of individuals who are susceptible to the disease, $E(t)$ represents the number of individuals who are exposed, $I(t)$ denotes the number of infectious individuals, Λ is the recruitment rate of the population, β the transmission rate or infection rate coeffcient, μ is the natural death of the population, σ is the rate at which exposed individuals become infectious, α is the death rate due to disease, $\delta \geq 0$ is the rate at which infectious individuals lose immunity and returns to susceptible class, and α_1 and α_2 are the parameter that measure the inhibitory effect.

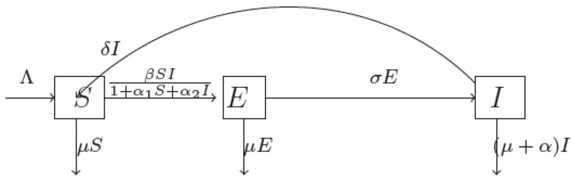

FIGURE 1. Sensitivity analysis of SEIS epidemic model parameters

The next part of this paper is organized as follows. In section 2, the positivity and boundedness of solutions as well as the formulation of the basic reproduction number. In section 3, we establish the existence of two positive equilibria. in section 4, we show that global asymptotic stability of the disease-free equilibrium depend only on the basic reproduction number. In section 5, we apply the geometric approach of Li and Muldowney to prove the global stability of endemic equilibrium. In section 6, we study the sensitivity analysis of the parameters. Numerical simulations are also presented in section 7 to illustrate the obtained results. Finally a brief discussion is given in section 8 to conclude this paper.

2. Model analysis

2.1. **Positivity and boundedness of solutions.** To begin, we establish the following proposition regarding the positivity and boundedness of the solutions of model 1.2.

Proposition 2.1. *All solutions of system 1.2 with nonnegative initial data remain nonnegative and bounded for all $t \geq 0$.*

Proof. The positivity of $S(t)$ is established for all $t \geq 0$. Suppose, for the sake of contradiction, that there exists a time $t_1 > 0$ where $S(t_1) = 0$. Substituting into the first equation of system 1.2, we find $S'(t_1) = A + \delta I(t_1) > 0$, which implies $S(t) < 0$ for $t \in (t_1 - \epsilon, t_1)$, where $\epsilon > 0$ is sufficiently small. This contradicts the assumption that $S(t) > 0$ for $t \in [0, t_1)$. Therefore, $S(t) > 0$ for all $t \geq 0$.

In similar fashion it can be shown that $E(t) > 0$ and $I(t) > 0$ for all $t \geq 0$.

Finally, to demonstrate the boundedness of the solutions, the total population is denoted by $N(t) = S(t) + E(t) + I(t)$, then we have

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \alpha I(t),$$

and $\limsup N(t) \leq N_0 = \frac{\Lambda}{\mu}$. We can study the three-dimensional system (1.2) in the following domain which is positively invariant:

$$T = \{(S, E, I) \in \mathbb{R}^3_+ : S + E + I \leq N_0\}.$$

$\square$

2.2. **The basic reproduction number.**

Theorem 2.1. *The basic reproduction number of model(1.2) is given by*

$$R_0 = \frac{\sigma\beta\Lambda}{(\mu+\sigma)(\mu+\alpha+\delta)(\mu+\alpha_1\Lambda)}.$$

Proof. The basic reproduction number is determined by means of the next-generation method see [20]. Writing the model (1.2) as follows

$$\frac{dx}{dt} = \mathcal{F} - \mathcal{V}$$

where $x = (E, I)^T$, and

$$\mathcal{F}(x) = \begin{bmatrix} \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I} \\ 0 \end{bmatrix},$$

and

$$\mathcal{V}(x) = \begin{bmatrix} (\mu+\sigma)E \\ -\sigma E + (\mu+\alpha+\delta)I \end{bmatrix}.$$

The jacobian matrix of $\mathcal{F}(x)$ and $\mathcal{V}(x)$ evaluated at $I = 0$, $E = 0$ and $S = \frac{\Lambda}{\mu}$ are respectively given by :

$$F = \begin{bmatrix} 0 & \frac{\beta\frac{\Lambda}{\mu}}{1+\alpha_1\frac{\Lambda}{\mu}} \\ 0 & 0 \end{bmatrix}.$$

and

$$V = \begin{bmatrix} \mu+\sigma & 0 \\ -\sigma & \mu+\alpha+\delta \end{bmatrix}.$$

The next-generation matrix is:

$$M = FV^{-1}.$$

where

$$V^{-1} = \frac{1}{\det(V)} \begin{bmatrix} \mu+\alpha+\delta & 0 \\ \sigma & \mu+\sigma \end{bmatrix},$$

and

$$\det(V) = (\mu+\sigma)(\mu+\alpha+\delta).$$

Thus,

$$V^{-1} = \frac{1}{(\mu+\sigma)(\mu+\alpha+\delta)} \begin{bmatrix} \mu+\alpha+\delta & 0 \\ \sigma & \mu+\sigma \end{bmatrix},$$

$$M = \frac{1}{(\mu+\sigma)(\mu+\alpha+\delta)} \begin{bmatrix} \frac{\beta\sigma\Lambda}{\mu+\alpha_1\Lambda} & \frac{\beta(\mu+\sigma)\Lambda}{\mu+\alpha_1\Lambda} \\ 0 & 0 \end{bmatrix}.$$

Finally, the dominant eigenvalue of M, which is R_0, is computed as follows:

$$R_0 = \frac{\beta\Lambda\sigma}{(\mu+\sigma)(\mu+\alpha+\delta)(\mu+\alpha_1\Lambda)}.$$

□

3. Existence of equilibria

The following theorem presents the existence and uniqueness of equilibria:

Theorem 3.1. *System (1.2) always has a disease-free equilibrium* $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ *which exists for all parameter values. On the other hand, if* $R_0 > 1$, *then system (1.2) also admits a unique endemic equilibrium:* $E_* = (S^*, E^*, I^*)$.

Proof. The steady state of model (1.2) S, E, I satisfying the following equations.

$$\begin{cases} \Lambda - \mu S - \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I} + \delta I = 0, \\ \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I} - (\mu+\sigma)E = 0, \\ \sigma E - (\mu+\alpha+\delta)I = 0. \end{cases} \tag{3.1}$$

If $I = 0$, we have $E = 0$, and $S = \frac{\Lambda}{\mu}$, therefore the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ of (1.2) exists.

If $I \neq 0$, we have

$$I^* = \frac{(\mu+\alpha_1\Lambda)(R_0-1)}{\mu\alpha_2 + (\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} - \delta)(\alpha_1(R_0-1) + \frac{R_0\mu}{\Lambda})},$$

$$E^* = \frac{(\mu+\alpha+\delta)I^*}{\sigma},$$

and

$$S^* = \frac{\Lambda - (\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} - \delta)I^*}{\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma}}.$$

Hence, there exist unique endemic equilibrium $P^* = (S^*, E^*, I^*)$ in the interior of T. This complete the proof. □

4. Global stability of the disease-free equilibrium

We have the following theorem on the global asymptotic stability of the disease-free equilibrium E_0 of (1.2.

Theorem 4.1.

(i) *If $R_0 \leq 1$, then the disease free equilibrium E_0 is globally asymptotically stable in T.*
(ii) *If $R_0 > 1$, then the disease free equilibrium E_0 is unstable.*

Proof. Set $V(t) = \sigma E(t) + (\mu+\sigma)I(t)$, then

$$\begin{aligned} V' &= \frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma}\sigma I(t)\Big(\frac{\beta S(t)}{1+\alpha_1 S(t)+\alpha_2 I(t)} - 1\Big) \\ &\leq \frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma}\sigma I(t)(R_0 - 1), \end{aligned}$$

We obtain that if $R_0 \leq 1$, then $V' \leq 0$. Furthermore, $V' = 0$ if and only if $I = 0$. Therefore the largest compact invariant set in $\{(S,E,I) \in T / V' = 0\}$, when $R_0 \leq 1$, is the singleton $\{E_0\}$. By LaSalle's Invariance Principle ([12], p.30) we conclude that E_0 is globally asymptotically stable in T. □

5. Stability analysis of the endemic equilibrium

5.1. **Local stability.** In this section, we prove the following theorem on the local asymptotic stability of the endemic equilibrium E^* of (1.2).

Theorem 5.1. *If $R_0 > 1$, then the endemic equilibrium E^* is locally asymptotically stable.*

Proof. Let $x = S - S^*$, $y = E - E^*$ and $z = I - I^*$. Then by linearizing system (1.2) around E^*, we have

$$\left\{ \begin{array}{l} \frac{dx}{dt} = -(\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2})x(t) - (\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} - \delta)z(t), \\ \frac{dy}{dt} = -(\mu+\sigma)y(t) + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2})x(t) + (\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} z(t)), \\ \frac{dz}{dt} = \sigma y(t) - (\mu+\alpha+\delta)z(t) \end{array} \right. \tag{5.1}$$

The characteristic equation associated to system (5.1)is given by

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \tag{5.2}$$

where

$$a_2 = 3\mu + \alpha + \sigma + \delta + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2},$$
$$a_1 = \sigma\Big(\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} - \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2}\Big)$$
$$+ (2\mu+\alpha+\delta+\sigma)\Big(\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2}\Big),$$
$$a_0 = \mu\sigma\Big(\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} - \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2}\Big)$$
$$+ \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2}\Big(\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} - \delta\Big)\sigma.$$

By the second and third equations in system (1.2), we have

$$\frac{(\mu+\sigma)(\mu+\alpha+\delta)}{\sigma} > \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^*)^2}. \tag{5.3}$$

By using (5.3), we can easily obtain that

$$a_i > 0, \qquad i = 0, 1, 2,$$

and

$$a_1 a_2 - a_0 > 0.$$

Hence, by the Routh-Hurwitz criterion, we have the local stability of P^* for $R_0 > 1$. This concludes the proof of Theorem 5.1. □

5.2. **Global stability.** In this section, we will use the geometric approach (see [13, 18]) to study the global stability of the endemic equilibrium P^*. sufficient conditions are obtained ensuring that P^* is globally asymptotically stable when $R_0 > 1$.

To show the existence of a compact set in the interior of T that is absorbing for (1.2) is equivalent to proving that (1.2)is uniformly persistent, which means that there exists a constant $\lambda > 0$ such that every solution $(S(t), E(t), I(t))$ of (1.2) with $(S(0), E(0), I(0))$ in the interior of T satisfies

$$\liminf_{t\to\infty} |(S(t), E(t), I(t))| \geq \lambda \tag{5.4}$$

Here λ is independent of initial data in T, see [13]. We can demonstrate the following result.

Proposition 5.1. *The system (1.2) is uniformly persistent if and only if $R_0 > 1$.*

Proof. By Theorem 4.3 in [9], we have that uniform persistence of system (1.2) is equivalent to instability of the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$. Combining the stability analysis for this equilibrium in Theorem 4.1 and Theorem 4.3 in [9], we know that system (1.2) is uniformly persistent if and only if $R_0 > 1$. □

Now, we have the following theorem about global stability of endemic equilibrium P^*.

Theorem 5.2. *Suppose that $R_0 > 1$. Then there exists $\overline{\delta} > 0$ such that the unique endemic equilibrium P^* is globally asymptotically stable when $\delta \leq \overline{\delta}$.*

Proof. By Proposition 5.1, when $R_0 > 1$, there exists a compact set $\mathcal{K}$ in the interior of T that is absorbing for (1.2). The proof of the Theorem consists of choosing a suitable vector norm in $\mathbb{R}^3$ and a 3×3 matrix-valued function $A(x)$ such that

$$\overline{q}_2 := \limsup_{t\to\infty} \sup_{x_0 \in \mathcal{K}} \frac{1}{t} \int_0^t \overline{\mu}(B(x(s, x_0)))ds < 0 \tag{5.5}$$

where $B = A_g A^{-1} + AJ^{[2]}A^{-1}$, $x = (S, E, I)$ and $f(x)$ denote the vector field of (1.2), i.e. $\frac{dx(t)}{dt} = f(x)$, A_f is obtained by replacing each entry a_{ij} of A by its derivative in the direction of f, and $\overline{\mu}(B)$ is the the Lozinski$\breve{i}$ measure of B with respect to the induced matrix norm.

The Jacobian matrix associated to (1.2) is given by:

$$J = \begin{pmatrix} -\mu - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & 0 & -\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} + \delta \\ \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & -\mu - \sigma & \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} \\ 0 & \sigma & -\mu - \alpha - \delta \end{pmatrix}.$$

The second additive compound matrix $J^{[2]}$ of the Jacobian matrix J is given by

$$J^{[2]} = \begin{pmatrix} -2\mu - \sigma - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} & \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta \\ \sigma & -2\mu - \alpha - \delta - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & 0 \\ 0 & \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & -2\mu - \alpha - \sigma - \delta \end{pmatrix}.$$

Consider the Lozinski$\breve{i}$ measure $\overline{\mu}$ of A with respect to a vector norm $\|.\|$ in $\mathbb{R}^{\binom{n}{2}}$, that is:

$$\overline{\mu} = \lim_{h\to 0^+} \frac{\|I + hA\| - 1}{h}.$$

Set the function $A(x) = A(S,E,I) = diag\{1, \frac{E}{I}, \frac{E}{I}\}$. Then,

$$A_f A^{-1} = diag\{0, \frac{E'}{E} - \frac{I'}{I}, \frac{E'}{E} - \frac{I'}{I}\},$$

where the matrix A_f is obtained by replacing each entry a_{ij} of $A(x)$ by its derivative in the direction of f. The matrix $B = A_f A^{-1} + AJ^{[2]}A^{-1}$ can be written in the following block form

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}, \tag{5.6}$$

where $B_{11} = -2\mu - \sigma - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2}$,

$$B_{12} = \frac{I}{E}\begin{pmatrix} \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} & \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta \end{pmatrix}, \qquad B_{21} = \begin{pmatrix} \frac{\sigma E}{I} \\ 0 \end{pmatrix}$$

$$B_{22} = \begin{pmatrix} \frac{E'}{E} - \frac{I'}{I} - 2\mu - \delta - \alpha - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & 0 \\ \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & \frac{E'}{E} - \frac{I'}{I} - 2\mu - \sigma - \alpha - \delta \end{pmatrix}$$

Let (u,v,w) denote the vectors in $\mathbb{R}^3 \cong \mathbb{R}^{\binom{3}{2}}$, we choose a norm in $\mathbb{R}^3$ as

$$|(u,v,w)| = \max\{|u|, |v| + |w|\},$$

and let $\overline{\mu}_1$ denote the Lozinski$\breve{i}$ measure with respect to norm $|.|$. Using the method of estimating $\overline{\mu}$ in [17], we have

$$\overline{\mu}(B) \leq \sup(g_1, g_2), \tag{5.7}$$

where

$$g_1 = B_{11} + |B_{12}|, \tag{5.8}$$

$$g_2 = \mu_1(B_{22}) + |B_{21}|, \tag{5.9}$$

where $\mu_1(B_{22})$ is the Lozinski$\breve{i}$ mesure of $2x2$ matrix B_{22} with respect to l_1 norm in $\mathbb{R}^2$, $|B_{12}|$ and $|B_{21}|$ are operators norms of B_{12} and B_{21}. We have

$$\mu_1(B_{11}) = -2\mu - \sigma - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2}, \tag{5.10}$$

$$|B_{21}| = \frac{\sigma E}{I}, \tag{5.11}$$

$$|B_{12}| = \frac{I}{E} \max\left\{ \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}, \left|\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta\right| \right\}, \tag{5.12}$$

and to calculate $\mu_1(B_{22})$, we add the absolute value of the off-diagonal one in each column of B_{22}, and then take the maximum of two sums, see ([6], p.41), We obtain:

$$\mu_1(B_{22}) = \frac{E'}{E} - \frac{I'}{I} - 2\mu - \delta - \alpha. \tag{5.13}$$

Then,

$$\begin{aligned} g_1 &= -2\mu - \sigma - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} \\ &\quad + \frac{I}{E} \max\left\{ \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}, \left|\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta\right| \right\}, \end{aligned} \tag{5.14}$$

$$g_2 = \frac{E'}{E} - \frac{I'}{I} - 2\mu - \delta - \alpha + \frac{\sigma . E}{I} \tag{5.15}$$

Rewriting (1.2) gives us

$$\frac{E'}{E} = \frac{\beta S I}{E(1+\alpha_1 S+\alpha_2 I)} - (\mu+\sigma), \tag{5.16}$$

$$\frac{I'}{I} = \frac{\sigma E}{I} - (\mu+\delta+\alpha). \tag{5.17}$$

Substituting equations 5.16 and 5.17 into 5.14 and 5.15 respectively, gives us,

$$\begin{aligned} g_1 &= \frac{E'}{E} - \mu - \frac{\beta S I}{E(1+\alpha_1 S+\alpha_2 I)} - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} \\ &\quad + \frac{I}{E} \max\left\{ \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}, \left|\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta\right| \right\}, \end{aligned} \tag{5.18}$$

and

$$g_2 = \frac{E'}{E} - \mu. \tag{5.19}$$

Since (1.2 is uniformly persistent when $R_0 > 1$ (see Proposition 5.1), there exists $\lambda > 0$ and $t_0 > 0$ such that $t > t_0$ implies

$$S(t) \geq \lambda, \quad E(t) \geq \lambda, \quad \text{and} \quad I(t) \geq \lambda,$$

for all $(S(0), E(0), I(0)) \in K$.

Set

$$\overline{\delta} := \inf\{\frac{2\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} : \lambda \leq S, I \leq \frac{A}{\mu}\} > 0,$$

If $\delta \leq \overline{\delta}$, then,

$$\max\left\{\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}, \left|\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} - \delta\right|\right\} = \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}$$

So, we can get easily

$$\begin{aligned} g_1 = \frac{E'}{E} - \mu - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} + \frac{\beta SI(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2 E} \\ - \frac{\beta SI}{E(1+\alpha_1 S+\alpha_2 I)} \leq \frac{E'}{E} - \mu. \end{aligned} \tag{5.20}$$

By 5.20 and 5.19, it is easy to show that

$$\overline{\mu}(B) \leq \frac{E'}{E} - \mu. \tag{5.21}$$

Thus, if $\delta \leq \overline{\delta}$, and for $t > t_0$ we have

$$\frac{1}{t}\int_0^t \overline{\mu}(B)dt \leq \frac{1}{t}\int_0^{t_0} \overline{\mu}(B)dt + \frac{1}{t}\log\frac{E(t)}{E(t_0)} - \mu\frac{t-t_0}{t} \leq \frac{-\mu}{2}, \tag{5.22}$$

and finally,

$$\overline{q}_2 < 0. \tag{5.23}$$

This concludes the proof. $\square$

6. Sensitivity Analysis

In this section, we perform the sensitivity analysis of R_0 to find ways to choose suitable parameters. Sensitivity indices measure the relative change in a state variable when a parameter changes. The normalized direct sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index can also be defined using partial derivatives (see [5]):

Definition 6.1. *The normalized forward sensitivity index of a variable, X, that depends differentiably on a parameter θ, is defined as:*

$$\Gamma_\theta^X = \frac{\partial X}{\partial \theta}.\frac{\theta}{X}.$$

A high sensitivity index indicates that a small change in the parameter θ produces a large change in X, which means that X is very sensitive to θ. Conversely, an index close to zero indicates low sensitivity.

Sensitivity indices play a fundamental role in model analysis and calibration, as they help identify the parameters that most influence the model's behavior and thus guide priorities for intervention or data collection measures.

A straightforward computation gives:

$$\Gamma_\sigma^{R_0} = \frac{\mu}{\mu+\sigma}, \quad \Gamma_\beta^{R_0} = 1, \quad \Gamma_\alpha^{R_0} = \frac{-\alpha}{\mu+\alpha+\delta}, \quad \Gamma_{\alpha_1}^{R_0} = \frac{-\alpha_1\Lambda}{\mu+\alpha_1\Lambda},$$

$$\Gamma_\mu^{R_0} = \frac{-\mu}{\mu+\sigma} - \frac{\mu}{\mu+\alpha_1 A} - \frac{\mu}{\mu+\alpha+\delta}, \quad \Gamma_\Lambda^{R_0} = \frac{\mu}{\mu+\alpha_1 A}, \quad \Gamma_\delta^{R_0} = \frac{-\delta}{\mu+\alpha+\delta}.$$

TABLE 1. Parameter values for Model 1.2

Parameter	Λ	β	σ	μ	α	α_1	δ
Value	100	0.05	0.04	0.02	0.01	0.02	0.01

TABLE 2. Sensitivity index table for Parameter values in TABLE 1

Parameter	**Index of Sensitivity**	**Order of sensitivity importance**
Λ	0.009	7
β	1	1
σ	0.333	4
μ	-0.843	3
α	-0.25	5
α_1	-0.990	2
δ	-0.25	5

By Table 2 and Fig. 6, the most sensitive parameters for the basic reproduction number R_0 are the transmission rate β, followed by α_1, then the natural death μ. The least sensitive parameter is the recruitment rate Λ.

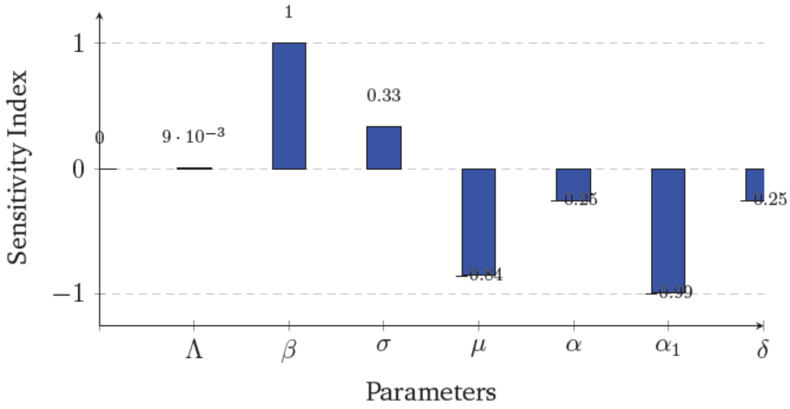

FIGURE 2. Sensitivity analysis of SEIS epidemic model parameters

7. NUMERICAL SIMULATIONS

In this section we perform numerical simulations to illustrate the the theoretical results obtained.

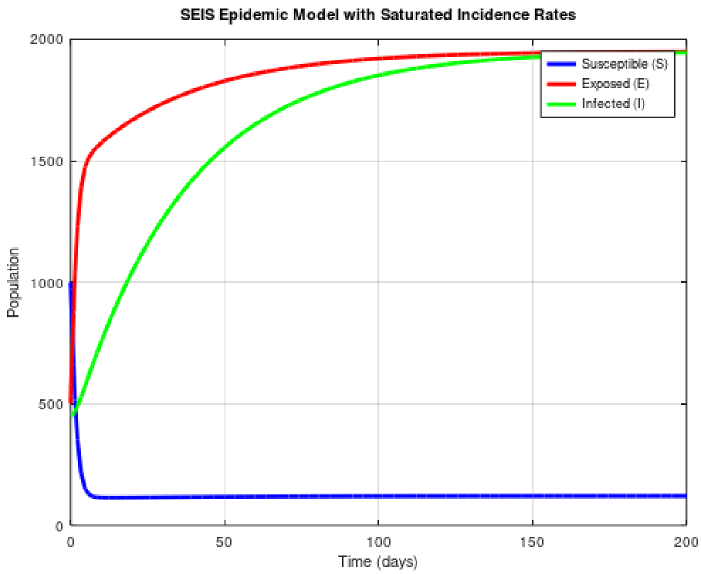

FIGURE 3. The dynamic behavior of compartiments S, E, and I in Model 1.2 with S(0)=1000, E(0)=500, I(0)=450, and Parameter values in TABLE 1. In this case $R_0 = 41.254 > 1$.

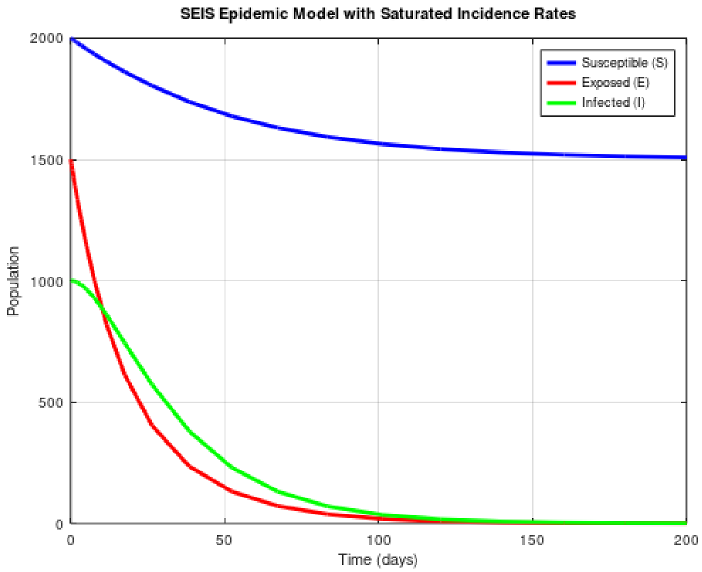

FIGURE 4. The dynamic behavior of compartiments S, E, and I in Model 1.2 with Parameter values in TABLE 3. In this case $R_0 = 0.0072 < 1$.

TABLE 3. Parameter values for Model 1.2

Parameter	Λ	β	σ	μ	α	α_1	δ	S(0)	E(0)	I(0)
Value	30	0.023	0.04	0.02	0.03	2	1	2000	1500	1000

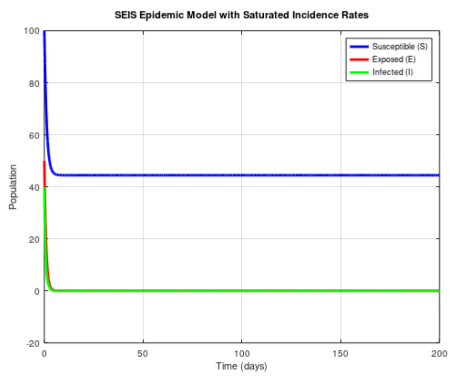

FIGURE 5. The dynamic behavior of compartiments S, E, and I in Model 1.2 with Parameter values in TABLE 4. In this case $R_0 = 0.155 < 1$.

TABLE 4. Parameter values for Model 1.2

Parameter	Λ	β	σ	μ	α	α_1	δ	S(0)	E(0)	I(0)
Value	40	0.5	0.7	0.9	0.9	0.5	0.5	100	50	40

TABLE 5. Parameter values for Model 1.2

Parameter	Λ	β	σ	μ	α	α_1	δ	S(0)	E(0)	I(0)
Value	0.45	2.5	0.07	0.09	0.09	0.95	0.05	10	5	4

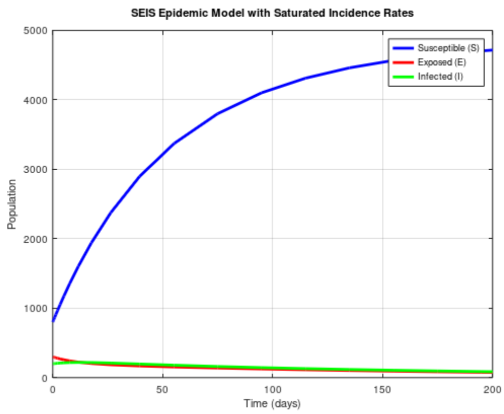

FIGURE 6. The dynamic behavior of compartiments S, E, and I in Model 1.2 with Parameter values in TABLE 5. In this case $R_0 = 3.871 > 1$.

8. DISCUSSION AND CONCLUSION

In summary, SEIS model with Beddington-DeAngelis infection rate provide a more realistic description of epidemic dynamics by accounting for the limited ability of an infectious disease to spread unchecked through a population. These models exhibit rich behaviors such as bifurcations and multiple equilibria, and their global dynamics are often governed by the basic reproduction number R_0. Understanding these dynamics helps in predicting the long-term behavior of an epidemic and in determining effective control strategies.

In this paper we formulated and analyzed an SEIS epidemic model with Beddington-DeAngelis infection rate (1.2). We have established the existence of two possible equilibrium points as well as their local and global stability. In Theorem 3.1, we prove that the model(1.2) always has a disease-free equilibrium $E_0 = (\frac{A}{\mu}, 0, 0)$ which exists for all parameter values. On the other hand, if the basic reproduction number R_0 is greater than unity, then model (1.2) also admits a unique endemic equilibrium $P* = (S^*, I^*, R^*)$. In Theorem 4.1 we prove that E_0 is globally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$. We prove in Theorem 5.1 that P^* is locally asymptotically stable. In Theorem 5.2, by means of the geometric approach we prove the global stability of the endemic equilibrium P^*.

REFERENCES

[1] A. ABTA, A. KADDAR, H. TALIBI ALAOUI: *Global stability for delay SIR and SEIR epidemic models with saturated incidence rates*, Electron. J. Diferential Equations, **2012** (23)(2012), 1–13.

[2] J.R. BEDDINGTON: *Mutual interference between parasites or predators and its effect on searching efficiency*, J. Anim. Ecol. , **44**(1) (1975), 331–340.

[3] F. BRAUER, C. CASTILLO-CHAVEZ: *Mathematical models in population biology and epidemiology*, vol. 2, Texts in Applied Mathematics, Springer, 2012.

[4] V. CAPASSO, G. SERIO : *A generalization of Kermack-Mckendrick deterministic epidemic model*, Math. Biosci., **42** (1978), 41–61.

[5] N. CHITNIS, J. M. HYMAN, J. M. CUSHING: *Determining important parameters in the spread of Malaria through the sensitivity analysis of a mathematical model* , Bulletin of Mathematical Biology, **70**(5) (2008), 1272–1296.

[6] W. A. COPPEL: *Stability and asymptotic behavior of differential equations*, Heath Boston, 1965.

[7] D. L. DEANGELIS, R. A. GOLDSTEIN, R. V. O'NEILL: *A model for tropic interaction Ecology*, **56**(4) (1975), 881–892.

[8] M. FAN, M. Y. LI, KE WANG: *Global stability of an SEIS epidemic model with recruitment and variyng total population size*, Mathematical Biosciences, **170** (2001), 199–202.

[9] H. I. FREEDMAN, M. X. TANG, AND S. G. RUAN: *Uniform persistence and flows near a closed positively invariant set*, J. Dynam. Differential Equations, **6** (1994), 583–600.

[10] A. KADDAR, A. ABTA, H. TALIBI ALAOUI: *A comparison of delayed SIR and SEIR epidemic models*, Nonlinear Analysis: Modelling and Control, **16**(2) (2011), 181–190.

[11] M. A. KHAN, Y. KHAN, S. ISLAM : *Complex dynamics of an SEIR epidemic model with saturated incidence rate and treatment*, Physica A: Statistical Mechanics and its Applications, **493**(2018), 210–227.

[12] Y.KUANG: *Delay differential equations with applications in population dynamics*, Vol. 191, Mathematics in Science and Engineering, Academic Press, Boston, 1993.

[13] M. Y. LI, J. S. MULDOWNEY : *A Geometric approch to global stability problems*, SIAM J. Math. Anal., **27**(4) (1996), 1070–1083.

[14] M. Y. LI, J. S. MULDOWNEY, P. V. D. DRIESSCHE : *Global stability of SEIRS models in epidemiology*, Canadian Applied Mathematics Quarterly, **7**(1999), 409–425.

[15] M. Y. Li, H. L. Smith, L. Wang : *Global dynamics of an SEIR epidemic model with vertical transmission*, SIAM J. Appl. Math., **62**(1) (2001), 58–69.

[16] F. Liu, F. Wei : *An epidemic model with Beddington-De Angelis functional response and environmental fluctuatons*, Physica A, **597** (2022), 1–15.

[17] R. H. Martin, Jr.: *Logarithmic norms and projections applied to linear differential systems*, Journal of Mathematical Analysis and Applications, **45** (1974), 432–454.

[18] R. A. Smith, : *Some applications of Hausdorff dimension inequalities for ordinary differential equations*, Proc. Roy. Soc. Edinburgh Sect. A, **104** (1986), 235–259.

[19] Swati. Nilam: *Fractional order SIR epidemic model with Beddington-DeAnglis incidence and Holling type II treatment rate for Covid-19*, Journal of Applied Mathematics and computing, **68** (2022), 3835–3859.

[20] P. van den Driessche, J. Watmough: *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Mathematical Biosciences, **180** (2002), 29–48.

[21] R. Xu, Z. Ma: *Stability of a delayed SIRS epidemic model with a nonlinear incidence rate*, Chaos, Solitons and Fractals, **41**(5) (2009), 2319–2325.

[22] R. Xu: *Global dynamics of an SEIS epidemic model with saturation incidence and latent period*, Applied Mathematics and Computation, **218**(5) (2012), 7927–7938.

[23] C. Wei, L. Chen: *A delayed epidemic model with pulse vaccination*, Discrete Dynamics in Nature and Society, **2008**, Article ID 746951, (2008), 1–12.

[24] J.-Z. Zhang, Z. Jin, Q.-X. Liu, Z.-Y. Zhang: *Analysis of a Delayed SIR Model with Nonlinear Incidence Rate*, Discrete Dynamics in Nature and Society, **2008**, Article ID 636153, (2008), 1–16.

Laboratory IMIS, Department of Mathematics and Computer Science
Faculty of Applied Sciences, National Road No. 10 city of Azrou, Ait Melloul
University of Ibn Zohr
Agadir,
Morocco.
Email address: s.elkhaiar@uiz.ac.ma