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Stability analysis of SIR and SIRS models with non monotone incidence function and various mortality rates

Yahya Mohamed[a], Aziza Ahmedou[b], Mohamed Saad Bouh Elemine Vall[b,*]

[a]*Quantitative Technics Department, Faculty of Legal and Economic Sciences, University of Nouakchott, Nouakchott, Mauritanie*

[b]*Department of Applied Mathematics and Industrial Engineering, Professional University Institute, University of Nouakchott, Nouakchott, Mauritanie*

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Abstract

This study employs the Lyapunov method, the Poincaré-Bendixson theorem, and the Dulac criterion to investigate the stability of SIR and SIRS models with non-monotone incidence and varying mortality rates. The analysis focuses on the stability properties of equilibrium points in the associated dynamical systems. For $R_0 < 1$, the eigenvalues of the Jacobian matrices at the equilibrium points have negative real parts, confirming their local asymptotic stability. When $R_0 > 1$, the global asymptotic stability of both the disease-free and endemic equilibrium points is demonstrated using a Lyapunov function and LaSalle's invariance principle. Additionally, an alternative approach leveraging the Poincaré-Bendixson theorem and Dulac's criterion is introduced to establish global stability. Numerical simulations, performed with carefully chosen parameters, validate the analytical results and provide deeper insights into the system's behavior.

Keywords: SIR epidemic model, Non monotone incidence rate, Global stability, Direct Lyapunov method, Dulac's criterion, Poincaré Bendixson theorem
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1 Introduction

Mathematical models became important tools for analyzing the spread and control of infectious diseases. The incidence function is an essential component to study the spread of infectious disease. In general the incidence is represented as a linear function of the infection class, by a principle of mass action:

$$g_1(I)S = \beta IS, \tag{1.1}$$

where S corresponds to the category of susceptibles, and I refers to the infected individuals, see for instance [2, 3, 4, 11, 12, 13] and the references therein.

In their study of cholera epidemic, Capasso and Serio [5] were the first to use a nonlinear incidence of Holling type II given by:

$$g_2(I)S = \frac{\beta IS}{1 + \alpha I}, \tag{1.2}$$

*Corresponding author
Email addresses: yahyajidou@yahoo.fr (Yahya Mohamed), ahmedouaziza@yahoo.fr (Aziza Ahmedou), saad2012bouh@gmail.com (Mohamed Saad Bouh Elemine Vall)

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where βI measure the infection force of the disease and $\frac{1}{1+\alpha I}$ measure the inhibition effect.
In [20] Liu et al. proposed a general incidence of the form:

$$g(I)S = \frac{\beta I^p S}{1+\alpha I^q}, \tag{1.3}$$

other authors have used this incidence see for example [6, 8, 10] for more generalised incidence of the form $f(S)g(I)$ and $f(S,I)$ we refer to [15, 21] .

If the function g is non-monotone, that is , g is increasing when I is small and decreasing when I is large, this incidence rate seems reasonable than the bi-linear incidence rate.

If the function g demonstrates non-monotonic behavior specifically, increasing at lower values of I and decreasing as I becomes larger it can provide insights into the psychological effects observed during an epidemic. When the number of infected individuals is very high, the force of infection may decline even as the infected population grows, as people often respond by limiting their social interactions to reduce exposure. The recent outbreak of Covid-19 illustrated this psychological impact on society. Measures such as border screenings, mask wearing, quarines, and isolations proved highly effective in curbing the infection rate during the later phases of the Covid-19 outbreak, even when the number of infected individuals was increasing see for example [8, 16, 17]. In addition, the global stability of SIR, SIRS epidemiological models with a non-linear function is strongly related to the concavity of the incidence function (see [15]). However, the incidence function considered in this study is not.To model this phenomenon, we propose a nonlinear and non-monotone incidence function of the previous form with $p = 1$ and $q = 2$, that is,

$$g(I)S = \frac{\beta IS}{1+\alpha I^2}, \tag{1.4}$$

such incidence is used in [25].

The global stability is an interesting classic question in the mathematical epidemiology models. Korobeinikov and Wake in [14] used a Lyapunov function of Volterra type to show the global stability of SIS, SIR and SIRS models with bi-linear incidence. Adda and Bichara have improved and completed Korobeinikov and Wake results by considering some SIR and SIRS models with bi-linear incidence and differential mortality rates [1]. By using the Poincaré-Bendixson theorem, the Dulac criteria, and the Lyapunov function method to establish the conditions for global stability, Vargas-De-Leon has studied the global stability of the SIS, SIR, and SIRS models with standard incidence in [23, 24]. More specifically, the author has constructed a Lyapunov function with two components, one of quadratic type and the second of Volterra type.

In this paper we propose two methods to show the global stability of SIR and SIRS models with nonmontone incidence rate and with various mortality rates. Our approach involves reducing the system to a planar one and then applying the Dulac criterion to eliminate periodic curves. To draw a conclusion, we also utilize the Poincaré-Bendixson theorem. Furthermore, we construct a Lyapunov function consisting of a quadratic term and a Volterra type term.

The paper is structured as follows: In the second section, we formulate the model and we calculate the basic reproduction number R_0 (It is defined as the number of new cases of infection caused by an infected individual in a population susceptible [7] [22]). In sections 3 and 4, we show that the disease-free equilibrium point is locally asymptotically stable if and only if $R_0 < 1$ and it is globally asymptotically stable. Finally when $R_0 > 1$ we show that the endemic equilibrium point is locally asymptotically stable and the global stability of endemic steady state using the Poincaré-Bendixson theorem, Dulac's criterion and Lyapunov's method in sections 5 and 6. In the section 7 we deduce similar results for the SIRS model. Numerical simulations were conducted to validate the analytical results presented in Section 8. Finally, we conclude in section 9.

2 Model presentation

In this section we describe an SIR model with various mortality rates and non monotone incidence function by assuming that the incubation period is negligible. Let $N(t)$ be total population which is divided into three classes: susceptible individuals $S(t)$, infected individuals $I(t)$, and individuals that have recovered/remove from the system $R(t)$. Based on this consideration, the total population is $N(t) = S(t) + I(t) + R(t)$.

We assume that all newborns become susceptible and that the births compensate the deaths, that is,

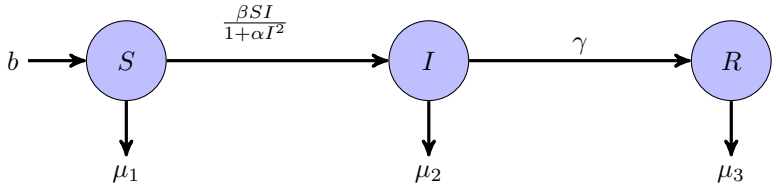
$$b = \mu_1 S + \mu_2 I + \mu_3 R. \tag{2.1}$$

This means the size of the population $N(t)$ remains constant. Here, μ_1, μ_2, and μ_3 indicate the mortality rates associated with the populations of susceptibles (S), infecteds (I), and recovereds (R).

We model the contact by the following non monotone function:

$$g(I) = \frac{\beta I}{1 + \alpha I^2},$$

with $\alpha > 0$, the terms βI and $1 + \alpha I^2$ represent respectively the force of the incidence and the inhibition effect. We have the following scheme:



Through the previous scheme, a system of nonlinear differential equations is obtained and presented below.

$$\begin{cases} S' = b - \mu_1 S - \frac{\beta SI}{(1+\alpha I^2)}, \\ I' = \frac{\beta SI}{(1+\alpha I^2)} - (\mu_2 + \gamma) I, \\ R' = \gamma I - \mu_3 R. \end{cases} \tag{2.2}$$

Based on (2.1) the previous system can be expressed as

$$\begin{cases} S' = -\frac{\beta SI}{(1+\alpha I^2)} + \mu_2 I + \mu_3 R, \\ I' = \frac{\beta SI}{(1+\alpha I^2)} - (\mu_2 + \gamma) I, \\ R' = \gamma I - \mu_3 R. \end{cases} \tag{2.3}$$

In the following table, we provide a detailed explanation of the parameters used in the previous system. Each parameter plays a crucial role in determining the behavior of the system.

Parameter	**Description**
b	Birth rate
β	Transmission rate.
α	Constant of saturation.
γ	Recovered rate.
μ_1, μ_2, μ_3	The death rates of susebtibles, infectes and recovered, respectively .

Table 1: Explanation of parameters in the control system model

Since $S(t) + I(t) + R(t) = N$=Constant, the system (2.3) is equivalent to the following planar system :

$$\begin{cases} S' = -\frac{\beta SI}{(1+\alpha I^2)} + \mu_2 I + \mu_3 (N - S - I), \\ I' = \frac{\beta SI}{(1+\alpha I^2)} - (\mu_2 + \gamma) I. \end{cases} \tag{2.4}$$

For the simplicity of notations, we put $\tilde{S} = \frac{S}{N}$, $\tilde{I} = \frac{I}{N}$, $\tilde{\beta} = \beta N$ and $\tilde{\alpha} = \alpha N^2$, which gives the following system

$$\begin{cases} \tilde{S}' = \mu_3 + (\mu_2 - \mu_3)\tilde{I} - \mu_3 \tilde{S} - \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2}, \\ \tilde{I}' = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2 + \gamma)\tilde{I}. \end{cases} \tag{2.5}$$

with $\tilde{S} \geq 0, \tilde{I} \geq 0$ such that $\tilde{S} + \tilde{I} \leq 1$. The biological domain of the previous system is the following standard compact positively invariant simplex given by

$$D = \left\{ (\tilde{S}, \tilde{I}) \in \mathbb{R}^2_+ : \tilde{S} + \tilde{I} \leq 1 \right\}.$$

3 Local stability of the disease-free equilibrium

It is very easy to show that the system (2.5) admits a unique disease-free equilibrium point given by $x_{DFE} = (\tilde{S}_0, 0) = (1, 0)$.
Furthermore, the basic reproduction number of the system (2.5) is obtained by Diekmann et al. in [7] and Driessche and Watmough in [22] as follows

$$R_0 = \frac{1}{\mu_2 + \gamma} \times \frac{\partial g}{\partial \tilde{I}}(1, 0) = \frac{\tilde{\beta}}{\mu_2 + \gamma}.$$

This enables us to state the following local stability result.

Theorem 3.1. The disease-free equilibrium point x_{DFE} is locally asymptotically stable if we assume that $R_0 < 1$ and unstable elsewhere.

Proof . This proof based on the direct calculation of eigenvalues of the Jacobian matrix of system, that is, the matrix defined by

$$J(\tilde{S}, \tilde{I}) = \begin{bmatrix} -\mu_3 - \frac{\tilde{\beta}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} & (\mu_2 - \mu_3) - \frac{\tilde{\beta}\tilde{S}(1-\tilde{\alpha}\tilde{I}^2)}{(1+\tilde{\alpha}\tilde{I}^2)^2} \\ \frac{\tilde{\beta}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} & \frac{\tilde{\beta}\tilde{S}(1-\tilde{\alpha}\tilde{I}^2)}{(1+\tilde{\alpha}\tilde{I}^2)^2} - (\mu_2 + \gamma) \end{bmatrix},$$

at the disease-free equilibrium point, we have

$$J(1, 0) = \begin{bmatrix} -\mu_3 & \mu_2 - \mu_3 - \tilde{\beta} \\ 0 & \tilde{\beta} - (\mu_2 + \gamma) \end{bmatrix}.$$

Therefore the eigenvalues of this matrix are $\lambda_1 = -\mu_3 < 0$ and $\lambda_2 = (\mu_2 + \gamma)(R_0 - 1)$.
Then, if $R_0 \geq 1$ the second eigenvalue λ_2 remains positive, this means that the disease-free equilibrium point x_{DFE} is unstable and if $R_0 < 1$, the second eigenvalue $\lambda_2 < 0$ then x_{DFE} is asymptotically stable. □

4 Global stability of disease-free equilibrium point

In order to establish the global stability of the disease-free equilibrium state, we develop a suitable Lyapunov function and use the LaSalle invariance principle, enabling us to examine the dynamics of system trajectories and their convergence to invariant sets associated with this Lyapunov function.

Theorem 4.1. Under the hypothesis $R_0 < 1$, the disease-free equilibrium point x_{DFE} is globally asymptotically stable.

Proof . Consider the following function, which represents the proportion of infected individuals and reflects the risk of disease transmission.

$$V(\tilde{S}, \tilde{I}) = \tilde{I}.$$

It is clear that this function can be viewed as a Lyapunov function and we have

$$\begin{aligned} V'(\tilde{S}, \tilde{I}) &= \tilde{I}', \\ &= \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1 + \tilde{\alpha}\tilde{I}^2} - (\mu_2 + \gamma)\tilde{I}, \\ &\leq (\mu_2 + \gamma)(R_0\tilde{S} - 1)\tilde{I}, \\ &\leq 0. \end{aligned}$$

If we assume that $V' = 0$ then $\tilde{I} = 0$ or ($\tilde{S} = \tilde{S}_0$ and $R_0 = 1$.) So the largest invariant set contained in the biological domain D is $M = \left\{(\tilde{S}, \tilde{I}) \in D : V' = 0\right\}$ which is reduced to the disease-free equilibrium point x_{DFE} and by the LaSalle invariance principle in [18, 19] the point x_{DFE} is globally asymptotically stable. □

5 Local stability of the endemic equilibrium point

We look at the existence of endemic equilibrium point. This endemic equilibrium state satisfies

$$\begin{cases} 0 = \mu_3 + (\mu_2 - \mu_3)\tilde{I} - \mu_3\tilde{S}^* - \frac{\tilde{\beta}\tilde{S}^*\tilde{I}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2}, \\ 0 = \frac{\tilde{\beta}\tilde{S}^*\tilde{I}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2} - (\mu_2 + \gamma)\tilde{I}^*. \end{cases}$$

By solving the previous nonlinear system, we will have

$$x^* = (\tilde{S}^*, \tilde{I}^*) = \left(\frac{1 + \tilde{\alpha}(\tilde{I}^*)^2}{R_0}, \quad \frac{-(\mu_3 + \gamma)R_0 + \sqrt{(\mu_3 + \gamma)^2 R_0^2 + 4\tilde{\alpha}\mu_3^2(R_0 - 1)}}{2\tilde{\alpha}\mu_3} \right).$$

It is easy to show that if $R_0 > 1$, then $\tilde{S}^* > 0, \tilde{I}^* > 0$ and $\tilde{S}^* + \tilde{I}^* \leq 1$. Now, we are in position to quote the following local stability theorem.

Theorem 5.1. If $R_0 > 1$. Then, the endemic equilibrium point x^* is locally asymptotically stable.

Proof . This proof is quite similar to the one in theorem 3.1. We start by giving the Jacobian matrix of system (2.5) at the endemic equilibrium, that is ,

$$J(\tilde{S}^*, \tilde{I}^*) = \begin{bmatrix} -\mu_3 - \frac{\tilde{\beta}\tilde{I}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2} & (\mu_2 - \mu_3) - \frac{\tilde{\beta}\tilde{S}^*(1-\tilde{\alpha}(\tilde{I}^*)^2)}{(1+\tilde{\alpha}(\tilde{I}^*)^2)^2} \\ \frac{\tilde{\beta}\tilde{I}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2} & \frac{\tilde{\beta}\tilde{S}^*(1-\tilde{\alpha}(\tilde{I}^*)^2)}{(1+\tilde{\alpha}(\tilde{I}^*)^2)^2} - (\mu_2 + \gamma) \end{bmatrix}.$$

Since at the endemic equilibrium point, we have

$$\frac{\tilde{\beta}\tilde{S}^*\tilde{I}^*}{1 + \tilde{\alpha}(\tilde{I}^*)^2} = (\mu_2 + \gamma)\tilde{I}^*,$$

which yields

$$\mu_2 + \gamma = \frac{\tilde{\beta}\tilde{S}^*}{1 + \tilde{\alpha}(\tilde{I}^*)^2},$$

and

$$\frac{\tilde{\beta}\tilde{S}^* - \tilde{\alpha}\tilde{\beta}\tilde{S}^*(\tilde{I}^*)^2}{\left(1 + \tilde{\alpha}(\tilde{I}^*)^2\right)^2} - (\mu_2 + \gamma) = \frac{\tilde{\beta}\tilde{S}^* - \tilde{\alpha}\tilde{\beta}\tilde{S}^*(\tilde{I}^*)^2}{\left(1 + \tilde{\alpha}(\tilde{I}^*)^2\right)^2} - \frac{\tilde{\beta}\tilde{S}^*\left(1 + \tilde{\alpha}(\tilde{I}^*)^2\right)}{\left(1 + \tilde{\alpha}(\tilde{I}^*)^2\right)^2} = \frac{-2\tilde{\alpha}\tilde{\beta}\tilde{S}^*(\tilde{I}^*)^2}{\left(1 + \tilde{\alpha}(\tilde{I}^*)^2\right)^2},$$

and because

$$\mu_2 - \mu_3 = \frac{\tilde{\beta}\tilde{S}^*}{1 + \tilde{\alpha}\tilde{I}^*} - \frac{\mu_3}{\tilde{I}^*}(1 - \tilde{S}^*).$$

The Jacobain matrix $J(x^*)$, can be written

$$J(x^*) = \begin{bmatrix} -\mu_3 - X & Y - \frac{\mu_3}{\tilde{I}^*}(1 - \tilde{S}^*) \\ X & -Y \end{bmatrix}.$$

where $X = \frac{\tilde{\beta}\tilde{I}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2}$, $Y = \frac{\tilde{\alpha}\tilde{\beta}\tilde{S}^*}{(\tilde{I}^*)^2 2\left(1+\tilde{\alpha}(\tilde{I}^*)^2\right)^2}$ and in consequences, we have

$$Tr(J(x^*)) = -\mu_3 - X - Y < 0 \quad \text{and} \quad \det J(x^*) = \mu_3 Y + \frac{\mu_3}{\tilde{I}^*}(1 - \tilde{S}^*)X \geq 0.$$

Thus, the eigenvalues of $J(x^*)$ have a negative real part, this means the endemic equilibrium x^* is locally asymptotically stable. □

6 Global stability of endemic equilibrium point

In this section, we propose two methods to prove the global stability result state in the following theorem.

6.1 Dulac's criteria

The Dulac criterion is a crucial approach in the analysis of stability within dynamical systems, offering a framework to assess the global stability of equilibria through the examination of vector field divergence. This criterion aids in understanding the behavior of trajectories near equilibria and evaluates whether solutions approach or move away from these points, thereby serving as an important tool for comprehending the dynamics of different systems.

We will apply this criterion to derive the following stability result.

Theorem 6.1. Assume that $R_0 > 1$. Then, the endemic equilibrium point is globally asymptotically stable.

Proof . Consider the following planar system

$$\begin{cases} \tilde{S}' = \mu_3 + (\mu_2 - \mu_3)\tilde{I} - \mu_3\tilde{S} - \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} = f_1(\tilde{S},\tilde{I}), \\ \tilde{I}' = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2+\gamma)\tilde{I} = f_2(\tilde{S},\tilde{I}). \end{cases}$$

We employ the Dulac's criterion to achieve this proof by taking the following Dulac function $B(\tilde{S},\tilde{I}) = \frac{1+\tilde{\alpha}\tilde{I}^2}{\tilde{\beta}\tilde{S}\tilde{I}}$, we obtain

$$\frac{\partial Bf_1}{\partial \tilde{S}}(\tilde{S},\tilde{I}) + \frac{\partial Bf_2}{\partial \tilde{I}}(\tilde{S},\tilde{I}) = -\frac{\mu_2(1+\tilde{\alpha}\tilde{I}^2)}{\tilde{\beta}\tilde{S}^2} - \frac{2\tilde{\alpha}\tilde{I}(\mu_2+\gamma)}{\tilde{\beta}\tilde{S}} + \frac{\mu_3(1+\tilde{\alpha}\tilde{I}^2)}{\tilde{\beta}\tilde{S}^2}(1-\frac{1}{\tilde{I}}) < 0.$$

Thus, the system (2.5) does not have a limit cycle in D.

In view of theorem 5.1 x^* is locally asymptotically stable and since D is positively invariant set, then Poincaré-Bendixson theorem shows that x^* is globally asymptotically stable. □

6.2 Lyapunov method

Now, we are in position to state another proof of Theorem 6.1 based on Lyapunov functions.

Proof . Let V be the Lyapunov function defined by

$$V(\tilde{S},\tilde{I}) = \frac{1}{2}(\tilde{S}-\tilde{S}^*+\tilde{I}-\tilde{I}^*)^2 + \frac{2\mu_3+\gamma}{\tilde{\beta}}(1+\tilde{\alpha}(\tilde{I}^*)^2)\left(\tilde{I}-\tilde{I}^*-\tilde{I}^*\ln\left(\frac{\tilde{I}}{\tilde{I}^*}\right)\right).$$

Then, the time derivative of V may be write as

$$\begin{aligned}
V'(\tilde{S},\tilde{I}) &= (\tilde{I}'+\tilde{S}')(\tilde{S}-\tilde{S}^*+\tilde{I}-\tilde{I}^*) + \frac{2\mu_3+\gamma}{\tilde{\beta}}(1+\tilde{\alpha}(\tilde{I}^*)^2)\tilde{I}'\left(1-\frac{\tilde{I}^*}{\tilde{I}}\right), \\
&= (\mu_3-\mu_3\tilde{S}-(\mu_3+\gamma)\tilde{I})(\tilde{S}-\tilde{S}^*+\tilde{I}-\tilde{I}^*) + \frac{2\mu_3+\gamma}{\tilde{\beta}}(1+\tilde{\alpha}(\tilde{I}^*)^2) \\
&\quad \left(\frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2+\gamma)\tilde{I}\right)\left(1-\frac{\tilde{I}^*}{\tilde{I}}\right), \\
&= (\mu_3(\tilde{S}^*-\tilde{S}) - (\mu_3+\gamma)(\tilde{I}^*-\tilde{I}))(\tilde{S}-\tilde{S}^*+\tilde{I}-\tilde{I}^*) \\
&\quad +(2\mu_3+\gamma)(1+\tilde{\alpha}(\tilde{I}^*)^2)\left(\frac{\tilde{S}}{1+\tilde{\alpha}\tilde{I}^2} - \frac{\tilde{S}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2}\right)\left(\tilde{I}-\tilde{I}^*\right), \\
&= -\mu_3(\tilde{S}-\tilde{S}^*)^2 - (\mu_3+\gamma)(\tilde{I}-\tilde{I}^*)^2 - (2\mu 3+\gamma)(\tilde{I}-\tilde{I}^*)(\tilde{S}-\tilde{S}^*), \\
&\quad +(2\mu_3+\gamma)(1+\tilde{\alpha}(\tilde{I}^*)^2)\left[\frac{\tilde{S}-\tilde{S}^*}{1+\tilde{\alpha}(\tilde{I}^*)^2} - \frac{\tilde{\alpha}\tilde{S}(\tilde{I}^*)^2-\tilde{I}^2)}{(1+\tilde{\alpha}\tilde{I}^2)(1+\tilde{\alpha}(\tilde{I}^*)^2)}\right](\tilde{I}-\tilde{I}^*), \\
&= -\mu_3(\tilde{S}-\tilde{S}^*)^2 - (\mu_3+\gamma)(\tilde{I}-\tilde{I}^*)^2 - \frac{(2\mu_3+\gamma)\tilde{\alpha}\tilde{S}(\tilde{I}^*+\tilde{I})}{1+\tilde{\alpha}\tilde{I}^2}(\tilde{I}-\tilde{I}^*)^2, \\
&\leq 0.
\end{aligned}$$

Therefore, $V' = 0$ if and only if $\tilde{S} = \tilde{S}^*$ and $\tilde{I} = \tilde{I}^*$. Thus the largest set $\{(\tilde{S},\tilde{I}) \in D : V' = 0\}$ is reduced to x^*.

Then, according to LaSalle principle in [18, 19] the positive equilibrium state x^* is globally asymptotically stable in D. □

7 SIRS Model Analysis

In this section, we examine a SIRS model that incorporates a non-monotonic incidence function and variable mortality rates. Retaining the notations from previous sections, we define the following system:

$$\begin{cases} S' = b - \mu_1 S - \frac{\beta SI}{1+\tilde{\alpha}I^2} + \rho R, \\ I' = \frac{\beta SI}{1+\tilde{\alpha}I^2} - (\mu_2 + \gamma)I, \\ R' = \gamma I - (\mu_3 + \rho)R, \end{cases} \tag{7.1}$$

which reduces to

$$\begin{cases} S' = -\frac{\beta SI}{1+\alpha I^2} + \mu_2 I + (\mu_3 + \rho)R, \\ I' = \frac{\beta SI}{1+\alpha I^2} - (\mu_2 + \gamma)I, \\ R' = \gamma I - (\mu_3 + \rho)R. \end{cases} \tag{7.2}$$

System (7.2) is structurally identical to system (2.3), with μ_3 replaced by $\mu_3 + \rho$. Introducing the scaled variables $\tilde{S} = \frac{S}{N}$, $\tilde{I} = \frac{I}{N}$, and $\tilde{R} = \frac{R}{N}$, system (7.2) can be reformulated as

$$\begin{cases} \tilde{S}' = -\frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} + \mu_2\tilde{I} + (\mu_3 + \rho)\tilde{R}, \\ \tilde{I}' = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2 + \gamma)\tilde{I}, \\ \tilde{R}' = \gamma\tilde{I} - (\mu_3 + \rho)\tilde{R}, \end{cases} \tag{7.3}$$

where $\tilde{\alpha} = (\alpha N)^2$ and $\tilde{\beta} = \beta N$. Given that $\tilde{S} + \tilde{I} + \tilde{R} = 1$, we can further simplify (7.3) as follows:

$$\begin{cases} \tilde{S}' = -\frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} + \mu_2\tilde{I} + (\mu_3 + \rho)(1 - \tilde{S} - \tilde{I}), \\ \tilde{I}' = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2 + \gamma)\tilde{I}, \end{cases} \tag{7.4}$$

which is equivalent to

$$\begin{cases} \tilde{S}' = (\mu_3 + \rho) - \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_3 + \rho)\tilde{S} + (\mu_2 - \rho - \mu_3)\tilde{I}, \\ \tilde{I}' = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{1+\tilde{\alpha}\tilde{I}^2} - (\mu_2 + \gamma)\tilde{I}. \end{cases} \tag{7.5}$$

The stability behavior of this final system is equivalent to that of system (2.5), with both systems exhibiting comparable stability characteristics.

8 Numerical simulation

Based on the parameter values presented in Table 2, which were carefully chosen to representatively illustrate the model dynamics, we performed numerical simulations using the R software. These simulations were carried out with the `ode()` function from the `deSolve` package, which by default implements the fourth-order Runge-Kutta method to solve the system of equations (2.2) describing the model. The primary goal of these simulations is to validate the analytical results obtained in Sections 4 and 6 by generating graphs that demonstrate that, when $R_0 < 1$, the disease-free equilibrium is achieved, with the infected populations progressively declining to extinction. Conversely, for $R_0 > 1$, an endemic equilibrium emerges, indicating the persistence of the disease within the population. These simulations also illustrate the effect of the saturation rate α on the variations of the susceptible and infected populations.

Table 2: The Value of all of parameters .

Parameter	Values	Source
b	1	assumed
β	0.3	assumed
α	0.5	assumed
γ	0.3	assumed
μ_1	0.01	assumed
μ_2	0.08	assumed
μ_3	0.05	assumed
ρ	0.05	assumed

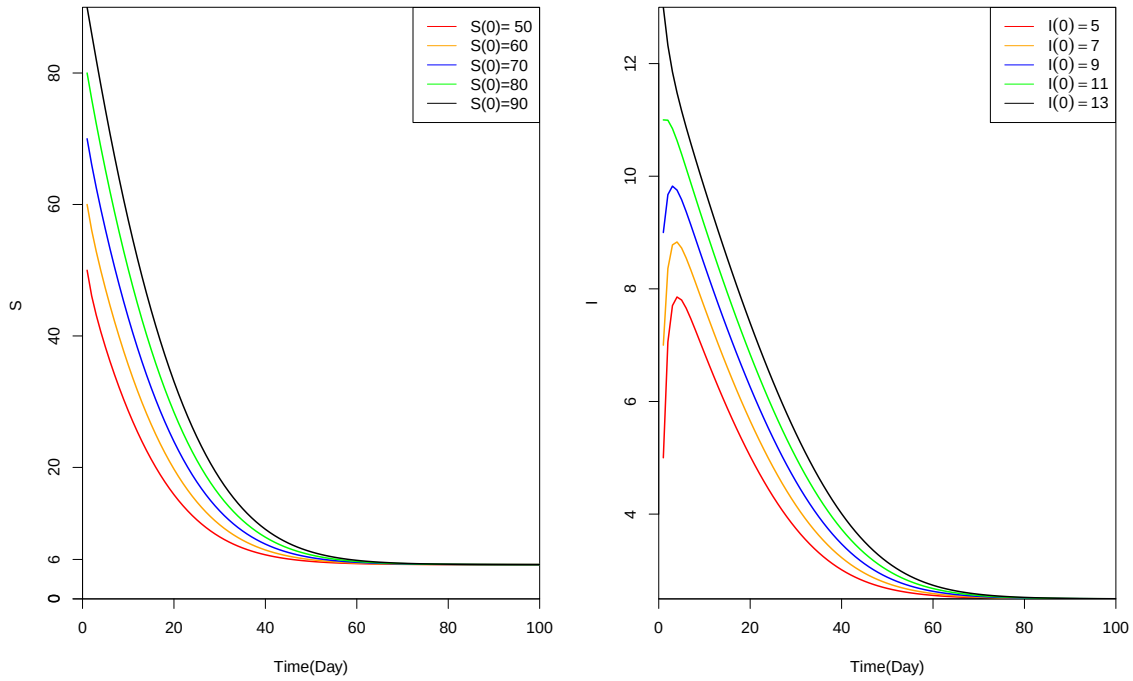


Figure 1: The plot of S and I when $R_0 = 0.78 < 1$ for different initial conditions and by using the values parameters from Table 2.

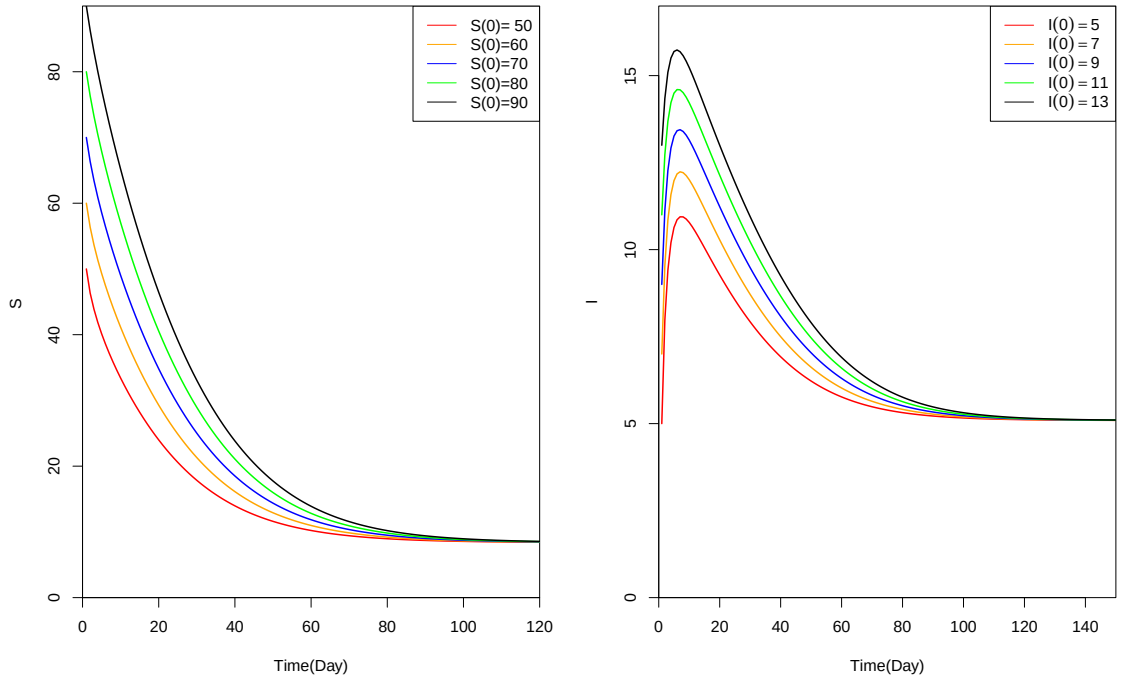


Figure 2: The plot of S and I when $R_0 = 1.6 > 1$ for various initial conditions based on the values parameters from Table 2 and with $\gamma = 0.1$.

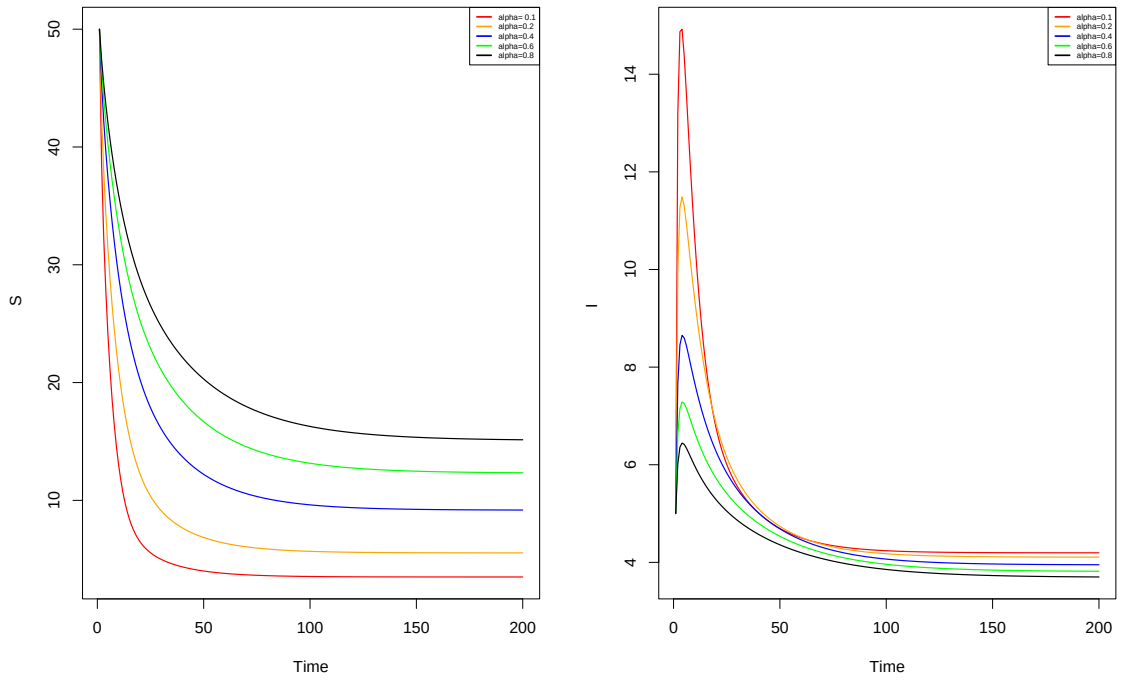


Figure 3: The plot of S and I for various value of α

Figure 1 illustrates the behavior of system (2.2) under different initial conditions when $R_0 = 0.78 < 1$. The results show that the susceptible population stabilizes at $S_0 = 5.2$ as $t \to \infty$, while the infected population gradually declines until it vanishes. Consequently, system (2.2) converges to the disease-free equilibrium $x_{DFE} = (5.2, 0)$ in accordance with Theorem 4.1. Furthermore, Figure 2 depicts the behavior of system (2.2) under various initial conditions when $R_0 = 1.6 > 1$. This indicate that susceptible and infected populations persist as $t \to \infty$, approaching the endemic equilibrium $x^* = (8.37, 5)$ as $t \to \infty$, thereby validating the conclusions of Theorem 6.1.

Finally, Figure 3 examines the effect of the saturation constant α on the susceptible and infected populations. It is observed that an increase in α leads to a rise in the susceptible population S, whereas a decrease in α results in an increase in the infected population I, thereby prolonging the duration of the epidemic and amplifying its severity.

9 Conclusion

In this paper, we consider an epidemic SIR model characterized by a non-monotonic incidence function defined as

$$f(I) = \frac{\beta SI}{1 + \alpha I^2},$$

where, βSI represents the infection force, describing the rate of new infections due to interactions between susceptible (S) and infected (I) individuals. The denominator $1 + \alpha I^2$ models an inhibitory effect, capturing factors such as behavioral changes or public health interventions that become more pronounced as the infection level rises. This function realistically depicts an epidemic scenario where the number of infections initially increases, reaches a peak, and subsequently declines, reflecting the natural or induced containment of the disease's spread over time [16].

In this study, we investigated the global stability of SIR and SIRS epidemiological models characterized by non-monotone incidence functions and varying mortality rates. Using a combination of mathematical tools, including the Poincaré-Bendixson theorem, Dulac's criterion, and Lyapunov functions, we rigorously proved conditions under which the disease-free and endemic equilibria are globally stable. Note that the global stability of epidemiological models with nonlinear general incidence function inherently relies on the concavity of the incidence function [15], but in this paper, the incidence considered does not exhibit this property. Our work generalizes and builds upon the results of Adda and Bichara in [1] and Korobeinikov and Wake in [14].

To further validate and illustrate the theoretical results, we performed detailed numerical simulations, which confirmed the predicted stability properties and dynamic behavior of the models.

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Declarations

Ethical Approval Statement

This research paper, does not require formal ethical approval as it does not involve human participants, sensitive personal data, or any potential ethical concerns.

Competing Interests Statement

These authors contributed equally to this work.

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The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

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