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Research article

Global investigation for an "SIS" model for COVID-19 epidemic with asymptomatic infection

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Abstract: In this paper, we analyse a dynamical system taking into account the asymptomatic infection and we consider optimal control strategies based on a regular network. We obtain basic mathematical results for the model without control. We compute the basic reproduction number ($\mathcal{R}$) by using the method of the next generation matrix then we analyse the local stability and global stability of the equilibria (disease-free equilibrium (DFE) and endemic equilibrium (EE)). We prove that DFE is LAS (locally asymptotically stable) when $\mathcal{R} < 1$ and it is unstable when $\mathcal{R} > 1$. Further, the existence, the uniqueness and the stability of EE is carried out. We deduce that when $\mathcal{R} > 1$, EE exists and is unique and it is LAS. By using generalized Bendixson-Dulac theorem, we prove that DFE is GAS (globally asymptotically stable) if $\mathcal{R} < 1$ and that the unique endemic equilibrium is globally asymptotically stable when $\mathcal{R} > 1$. Later, by using Pontryagin's maximum principle, we propose several reasonable optimal control strategies to the control and the prevention of the disease. We mathematically formulate these strategies. The unique optimal solution was expressed using adjoint variables. A particular numerical scheme was applied to solve the control problem. Finally, several numerical simulations that validate the obtained results were presented.

Keywords: COVID-19; SIS epidemic model; asymptomatic and symptomatic individuals; nonlinear incidence rates; Bendixson-Dulac theorem; optimal strategies; sensitivity analysis

1. Introduction

COVID-19 is one of the three Coronaviruses that has caused epidemic outbreaks over the last two decades. It can spread through close contact, coughing, sneezing, or talking. For COVID-19, there are two types of infected individuals: one is symptomatic infected individuals, defined as those who show symptoms (such as fever, cough, sore throat, etc.) after obtaining infected. The other is asymptomatic infected individuals, defined as those who do not show symptoms after obtaining infected [1, 2].

Asymptomatic cases are not confirmed cases which are divided into two different states. The first one is the asymptomatic individuals who show no symptoms for the whole time of the infection. The second one is the asymptomatic individuals who exhibit symptoms after a period of the asymptomatic infection. In the generally tested subgroup, the proportion of asymptomatic individuals who are found to be positive for COVID-19 is alarmingly high. Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) shows that asymptomatic and mildly symptomatic infections may be the key to disease transmission [3, 4]. Most asymptomatic infected individuals do not seek medical help because they have no obvious clinical signs which leads to the rapid spread of the disease. Therefore, the prevention and control of this specific type of patient on a global scale is a huge challenge and requires more attention from the world.

The global health crisis of the Coronavirus Covid-19 has brought to light the role of mathematical modeling in political and health decision-making [5–8]. We will study the SIS model inspired by the current Coronavirus outbreak. Mathematical models of infectious diseases have been used for over a decade to study and understand the mechanism of disease spread, predict the future of epidemics, and determine the best treatment strategy to protect human health. The famous SIR and SEIR mathematical epidemic models are the most used models providing good descriptions of infectious diseases especially when taking account of either symptomatic and asymptomatic infectious or time delay [9–12].

In this work, we have designed a compartmental ODE model to investigate the COVID-19 spreading dynamics incorporating non-linear saturated incidence rates for both asymptomatic and symptomatic infection. The incidence rate is an essential component for infectious disease transmission in a mathematical model for both symptomatic and asymptomatic infectious [13]. In the epidemiology perception, incidence rate measures the number of new cases of a disease within a specified time duration as a percentage of the number of persons at threat for the disease [14]. We have also included two control functions designing quarantine strategies which is used to reduce the contact among infected individuals. Here we have studied the qualitative nature of the model through investigation of local and global nature of equilibrium points. Finally we have characterised optimal control to reduce the outbreak size and the control implementation costs. Results of the optimal control model show that optimal control provides significant reduction in COVID-19 spread.

The paper is organised as follows: In Section 2, we have formulated the proposed model and have studied the boundedness and positivity of solutions. Steady state analysis with constant controls have been done in Section 3 with respect to the basic reproduction number ($\mathcal{R}$). In Section 4, we characterised the optimal control model and we give an efficiency analysis. Finally, some numerical simulations and discussions were given in Section 5. The conclusions of our study has been reported in Section 6.

2. Model development

In this paper, we have studied a compartmental ODE model to investigate the COVID-19 spreading dynamics using non-linear saturated incidences. For this purpose we have formulated an ODE model by splitting the total host population Λ into three disjoint epidemiological classes specifically susceptible individuals $S(t)$, asymptomatic infectious individuals $I_a(t)$ and symptomatic infectious individuals $I_s(t)$ i.e. $\Lambda = S(t) + I_a(t) + I_s(t)$.

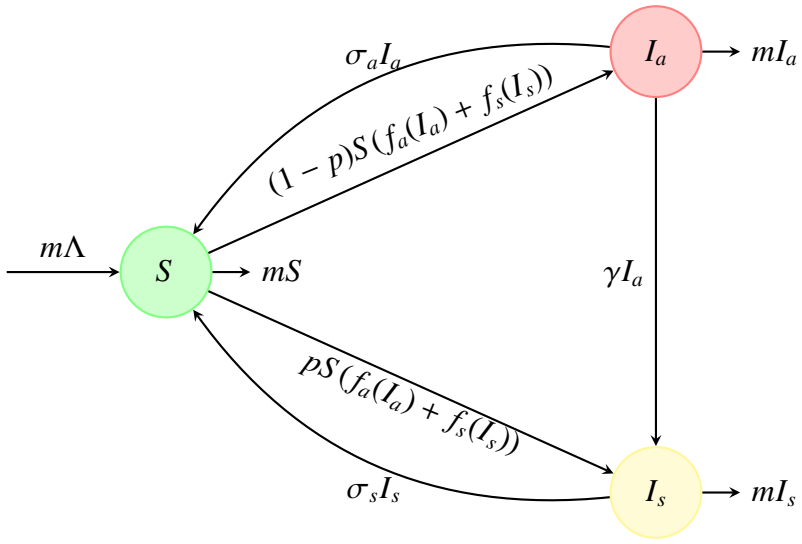

Figure 1. Dynamical system taking into account the asymptomatic infection. Circles describe the compartments S, I_a and I_s and the arrows (and labels) correspond to rates of transition between them.

To formulate the model we have considered the following factors:

- Susceptible individual once infected has a probability $0 < p < 1$ to be symptomatic and then a probability $0 < 1 - p < 1$ to be asymptomatic.
- Susceptible individual is infected by asymptomatic infected individuals at a nonlinear increasing rate $f_a(I_a)S$ and by symptomatic infected individuals at a nonlinear increasing rate $f_s(I_s)S$. The importance of these increasing incidence rates is that the rate of effective contacts between infected individuals and susceptible individuals increases with the increase of number of infective individuals.
- asymptomatic and symptomatic individuals are recovered at constant rates σ_a and σ_s, respectively.
- Recovered individuals can catch the diseases and then they are added to susceptible compartment.
- asymptomatic individuals become symptomatic individuals at a constant rate γ.
- Susceptible individuals are recruited at constant rate $m\Lambda$, natural mortality rate is m.

According to the epidemiological assumptions, we proposed the following mathematical model

$$\begin{cases} \dot{S} &= m\Lambda - S(f_a(I_a) + f_s(I_s)) + \sigma_a I_a + \sigma_s I_s - mS, \\ \dot{I}_a &= (1-p)S(f_a(I_a) + f_s(I_s)) - \gamma I_a - mI_a - \sigma_a I_a, \\ \dot{I}_s &= p\,S(f_a(I_a) + f_s(I_s)) + \gamma I_a - mI_s - \sigma_s I_s, \end{cases} \tag{2.1}$$

with positive initial condition $(S^0, I_a^0, I_s^0) \in \mathbb{R}^3_+$.

3. Mathematical analysis

Assumption 1. *f_a and f_s are bounded, non-negative $C^1(\mathbb{R}_+)$, concave and increasing functions with $f_a(0) = f_s(0) = 0$.*

Table 1. Parameters and variables of system (3.2).

	Notation	Definition
State variables	$S(t)$	Number of susceptible individuals at time t
	$I_a(t)$	Number of asymptomatic infected individuals at time t
	$I_s(t)$	Number of symptomatic infected individuals at time t
Functions	f_a	Saturated incidence rate in the asymptomatic compartment
	f_s	Saturated incidence rate in the symptomatic compartment
Parameters	Λ	Recruitment rate
	γ	Rate at which an individual enters the symptomatic compartment from the asymptomatic compartment.
	$1/\gamma$	Duration time spent in compartment I_a
	σ_a	Per-capita recovery rate of the infected individual in the asymptomatic compartment
	σ_s	Per-capita recovery rate of the infected individual in the symptomatic compartment
	m	Per-capita natural mortality rate
	$0 < p < 1$	Probability at which those newly infected individuals enter the symptomatic compartment
	$(1-p)$	Probability at which those newly infected individuals enter the asymptomatic compartment

Lemma 1. *The general non-linear incidence rates f_a and f_s satisfy $f_a'(I)I \leq f_a(I) \leq f_a'(0)I$ and $f_s'(I)I \leq f_s(I) \leq f_s'(0)I$, $\forall I > 0$.*

Proof. Let $I, I_1 \in \mathbb{R}_+$, and the function $g_1(I) = f_a(I) - If_a'(I)$. Since $f_a'(I) \geq 0$ (f_a is increasing function) and $f_a''(I) \leq 0$ (f_a is concave) then $g_1'(I) = -If_a''(I) \geq 0$ and $g_1(I) \geq g_1(0) = 0$. Therefore, $f_a(I) \geq If_a'(I)$. Similarly, let $g_2(I) = f_a(I) - If_a'(0)$ then $g_2'(I) = f_a'(I) - f_a'(0) \leq 0$ once f_a is a concave function. Thus $g_2(I) \leq g_2(0) = 0$ and $f_a(I) \leq If_a'(0)$. Similarly for the function f_s. □

It is necessary that the state variables $S(t), I_a(t)$ and $I_s(t)$ remain non-negative for all $t \geq 0$.

Proposition 1. $\Omega_0 = \left\{(S, I_a, I_s) \in \mathbb{R}_+^3 \ / \ S + I_a + I_s = \Lambda\right\}$ *is a positively invariant compact set for model (2.1).*

Proof. Assume that the initial condition $(S^0, I_a^0, I_s^0) \in \mathbb{R}_+^3$. If $S = 0$ then $\dot{S} = m\Lambda + \sigma_a I_a + \sigma_s I_s > 0$ therefore $S(t) > 0$ for all $t > 0$. Similarly, assume that $I_a = 0$ then $\dot{I}_a = (1-p)Sf_s(I_s) \geq 0$ therefore $I_a(t) \geq 0$ for all $t > 0$. By the same way, assume that $I_s = 0$ therefore $\dot{I}_s = pSf_a(I_a) + \gamma I_a \geq 0$ then $I_s(t) \geq 0$ for all $t > 0$. Therefore the model (3.2) admits a non-negative solution.
By summing the equations of (2.1), we get, for $T = S + I_a + I_s - \Lambda$, the following equation :

$$\dot{T} = \dot{S} + \dot{I}_a + \dot{I}_s = m\Lambda - mS - mI_a - mI_s = -mT.$$

Hence

$$T(t) = T(0)e^{-mt}. \tag{3.1}$$

Hence, Ω_0 is invariant for the model (2.1) due to all variables are non-negative. □

Using the conservation principles, we can compute S as function of I_a and I_s :

$$S = \Lambda - I_a - I_s.$$

Now, we can reduce the analysis of the original system (2.1) to the analysis of the following two-dimensional limiting system on the invariant set Ω :

$$\begin{cases} \dot{I}_a &= (1-p)(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) - \gamma I_a - mI_a - \sigma_a I_a, \\ \dot{I}_s &= p(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) + \gamma I_a - mI_s - \sigma_s I_s, \end{cases} \tag{3.2}$$

with positive initial condition $(I_a^0, I_s^0) \in \mathbb{R}_+^2$.
$\Omega = \left\{(I_a, I_s) \in \mathbb{R}_+^2 \ / \ I_a + I_s \leq \Lambda\right\}$ is a positively invariant compact set for model (3.2). It is obvious that $E_0 = (0, 0)$ is the only disease-free equilibrium of system (3.2).

The global behavior of our system inevitably depends on the basic reproduction number ($\mathcal{R}$), that is, the average number of secondary cases produced by an infectious individual who is introduced into an established population only of susceptible.

To drive the basic reproduction number ($\mathcal{R}$) for complex compartmental models, we use the next-generation operator approach proposed by Diekmann et al. [15,16]. Following the approach of van den Driessche and Watmough, we can rewrite (3.2)

$$\begin{pmatrix} \dot{I}_a \\ \dot{I}_s \end{pmatrix} = \begin{pmatrix} (1-p)(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) \\ p(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) \end{pmatrix} - \begin{pmatrix} (\gamma + m + \sigma_a)I_a \\ -\gamma I_a + (m + \sigma_s)I_s \end{pmatrix} = \mathcal{F} - \mathcal{V} \tag{3.3}$$

where $\mathcal{F}$ denotes the rate of appearance of new infections, and $\mathcal{V}$ denotes the rate of transfer of individuals into or out of each population set. Furthermore,

$$F = D\mathcal{F}(E_0) = \begin{pmatrix} (1-p)\Lambda f_a'(0) & (1-p)\Lambda f_s'(0) \\ p\Lambda f_a'(0) & p\Lambda f_s'(0) \end{pmatrix}, V = D\mathcal{V}(E_0) = \begin{pmatrix} (\gamma + m + \sigma_a) & 0 \\ -\gamma & (m + \sigma_s) \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \dfrac{1}{(\gamma + m + \sigma_a)} & 0 \\ \gamma & \dfrac{1}{(m + \sigma_s)} \end{pmatrix}, FV^{-1} = \Lambda \begin{pmatrix} \dfrac{(1-p)f_a'(0)}{(\gamma + m + \sigma_a)} + \gamma(1-p)f_s'(0) & \dfrac{(1-p)f_s'(0)}{(m + \sigma_s)} \\ \dfrac{pf_a'(0)}{(\gamma + m + \sigma_a)} + \gamma p f_s'(0) & \dfrac{pf_s'(0)}{(m + \sigma_s)} \end{pmatrix}.$$

Since $\det(FV^{-1}) = 0$, then one of the eigenvalues is zero. Therefore, we can deduce the second eigenvalue since their sum is equal to the trace of the matrix FV^{-1}. Then, the basic reproduction number which is the spectral radius of FV^{-1} (the maximum of the absolute values of its eigenvalues) is given by

$$\begin{aligned} \mathcal{R} &= \frac{(1-p)\Lambda f_a'(0)}{(\gamma + m + \sigma_a)} + \gamma(1-p)\Lambda f_s'(0) + \frac{p\Lambda f_s'(0)}{(m + \sigma_s)} \\ &= \frac{(1-p)\Lambda f_a'(0)}{(\gamma + m + \sigma_a)} + \frac{(p + \gamma(1-p)(m + \sigma_s))\Lambda}{(m + \sigma_s)} f_s'(0) \\ &\triangleq \mathcal{R}_1 + \mathcal{R}_2. \end{aligned} \tag{3.4}$$

Proposition 2. *The model (3.2) admits a unique disease-free equilibrium $E_0 = (0, 0)$ and a unique endemic equilibrium $E^* = (I_a^*, I_s^*)$ such that $I_a^*, I_s^* > 0$.*

Proof. Equilibria of (3.2) satisfy

$$\begin{cases} 0 &= (1-p)(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) - \gamma I_a - mI_a - \sigma_a I_a, \\ 0 &= p(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) + \gamma I_a - mI_s - \sigma_s I_s, \end{cases} \tag{3.5}$$

which reduces to

$$(\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) = \frac{(\gamma + m + \sigma_a)I_a}{(1-p)} = \frac{-\gamma I_a + (m+\sigma_s)I_s}{p}, \tag{3.6}$$

Thus

$$\begin{cases} I_s = \dfrac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a \\ (\Lambda - I_a - I_s)(f_a(I_a) + f_s(I_s)) = \dfrac{(\gamma + m + \sigma_a)I_a}{(1-p)}, \end{cases} \tag{3.7}$$

We conclude that from (3.6)

$$\left(\Lambda - I_a - \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a\right)\left(f_a(I_a) + f_s\left(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a\right)\right) = \frac{(\gamma + m + \sigma_a)I_a}{(1-p)}.$$

We can write this equation on the form

$$I_a g(I_a) = 0$$

where the function g given by

$$g(I_a) = \left(\Lambda - I_a - \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a\right)\left(\frac{f_a(I_a)}{I_a} + \frac{f_s\left(\dfrac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a\right)}{I_a}\right) - \frac{(\gamma + m + \sigma_a)}{(1-p)}.$$

- If $I_a = 0$, then $I_s = 0$. This equilibrium named the disease-free equilibrium will be noted here by $E_0 = (0, 0)$.
- If $I_a \neq 0$, then $g(I_a) = 0$. Let us calculate the derivative of the function g given by

$$g'(I_a) = -(1 + \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a)(\frac{f_a(I_a)}{I_a} + \frac{f_s(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a)}{I_a}) + (\Lambda - I_a - \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a)$$
$$(\frac{f_a'(I_a)I_a - f_a(I_a)}{I_a^2} + \frac{f_s'(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a) - f_s(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)} I_a)I_a}{I_a^2}).$$

The functions f_a and f_s satisfy $f_a'(I)I \leq f_a(I)$ and $f_s'(I)I \leq f_s(I)$, $\forall I \geq 0$ and all variables are non-negative, hence g is an increasing function and satisfies $g'(I_a) < 0$.

An easy calculation gives

$$\begin{aligned}\lim_{I_a\to 0} g(I_a) &= \lim_{I_a\to 0}(\Lambda - I_a - \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}I_a)(\frac{f_a(I_a)}{I_a} + \frac{f_s\left(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}I_a\right)}{I_a}) - \frac{(\gamma+m+\sigma_a)}{(1-p)}\\ &= \Lambda\left(f_a'(0) + \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}f_s'(0)\right) - \frac{(\gamma+m+\sigma_a)}{(1-p)}\\ &= \frac{(\gamma+m+\sigma_a)}{(1-p)}\left(\frac{\Lambda(1-p)}{(\gamma+m+\sigma_a)}f_a'(0) + \frac{\Lambda(\gamma+p(m+\sigma_a))}{(\gamma+m+\sigma_a)(m+\sigma_s)}f_s'(0) - 1\right)\\ &= \frac{(\gamma+m+\sigma_a)}{(1-p)}(\mathcal{R}-1),\end{aligned}$$

and

$$g(\Lambda) = -\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}\Lambda\left(\frac{f_a(\Lambda)}{\Lambda} + \frac{f_s\left(\frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}\Lambda\right)}{\Lambda}\right) - \frac{(\gamma+m+\sigma_a)}{(1-p)} < 0.$$

Since $\mathcal{R} > 1$, $g(\Lambda) < 0$ and $\lim_{I_a\to 0} g(I_a) < 0$, then $g(I_a) = 0$ has a unique positive solution I_a^* in $(0, \Lambda)$ and therefore the equilibrium state $E^* = (I_a^*, I_s^*)$ is unique with $I_s^* = \frac{\gamma + p(m+\sigma_a)}{(1-p)(m+\sigma_s)}I_a^*$.

□

3.1. Local analysis

In this subsection, the local stability behaviours of equilibria are discussed.

Theorem 1. *E_0 is LAS when $\mathcal{R} < 1$ and it is unstable when $\mathcal{R} > 1$.*

Proof. The Jacobian matrix given at the equilibrium point E_0 is

$$J_0 = \begin{pmatrix} (1-p)\Lambda f_a'(0) - (\gamma+m+\sigma_a) & (1-p)\Lambda f_s'(0) \\ p\Lambda f_a'(0) + \gamma & p\Lambda f_s'(0) - (m+\sigma_s) \end{pmatrix}.$$

The trace is given by

$$\text{Trace}\,(J_0) = (1-p)\Lambda f_a'(0) + p\Lambda f_s'(0) - (\gamma+m+\sigma_a) - (m+\sigma_s).$$

Since $\mathcal{R} = \frac{p\Lambda f_s'(0)}{(m+\sigma_s)} + \frac{(1-p)\Lambda f_a'(0)}{(\gamma+m+\sigma_a)} + \frac{(1-p)\gamma\Lambda f_s'(0)}{(\gamma+m+\sigma_a)(m+\sigma_s)}$ then when $\mathcal{R} < 1$, it is obvious that $\frac{p\Lambda f_s'(0)}{(m+\sigma_s)} < 1$, $\frac{(1-p)\Lambda f_a'(0)}{(\gamma+m+\sigma_a)} < 1$ therefore Trace $(J_0) < 0$. The determinant is given by

$$\begin{aligned}\text{Det}\,(J_0) &= \left((1-p)\Lambda f_a'(0) - (\gamma+m+\sigma_a)\right)\left(p\Lambda f_s'(0) - (m+\sigma_s)\right) - (1-p)\Lambda f_s'(0)\left(p\Lambda f_a'(0) + \gamma\right)\\ &= -p(\gamma+m+\sigma_a)\Lambda f_s'(0) + (\gamma+m+\sigma_a)(m+\sigma_s) - (1-p)(m+\sigma_s)\Lambda f_a'(0) - (1-p)\gamma\Lambda f_s'(0)\\ &= (\gamma+m+\sigma_a)(m+\sigma_s)\left(1 - \frac{p\Lambda f_s'(0)}{(m+\sigma_s)} - \frac{(1-p)\Lambda f_a'(0)}{(\gamma+m+\sigma_a)} - \frac{(1-p)\gamma\Lambda f_s'(0)}{(\gamma+m+\sigma_a)(m+\sigma_s)}\right)\\ &= (\gamma+m+\sigma_a)(m+\sigma_s)(1-\mathcal{R}) > 0 \text{ if } \mathcal{R} > 1.\end{aligned}$$

If $\mathcal{R} < 1$, then we have negative eigenvalues. Therefore, E_0 is LAS. Whereas, if $\mathcal{R} > 1$, E_0 is therefore unstable. □

Theorem 2. *E^* is LAS when $\mathcal{R} > 1$.*

Proof. For the equilibrium point E^*, the Jacobian is given by $J^* = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix}$ where

$$\begin{aligned}
a_{11} &= -(1-p)(f_a(I_a^*)+f_s(I_s^*)) + (1-p)(\Lambda - I_a^* - I_s^*)f_a'(I_a^*) \; - \; (\gamma + m + \sigma_a) \\
&= -(1-p)(f_a(I_a^*)+f_s(I_s^*)) + (1-p)(\Lambda - I_a^* - I_s^*)f_a'(I_a^*) - (1-p)(\Lambda - I_a^* - I_s^*)\frac{(f_a(I_a^*)+f_s(I_s^*))}{I_a^*} \\
&= -(1-p)(f_a(I_a^*)+f_s(I_s^*)) + (1-p)(\Lambda - I_a^* - I_s^*)\left(f_a'(I_a^*) - \frac{f_a(I_a^*)}{I_a^*}\right) - (1-p)(\Lambda - I_a^* - I_s^*)\frac{f_s(I_s^*)}{I_a^*} \\
&\leq -(1-p)(f_a(I_a^*)+f_s(I_s^*)) - (1-p)(\Lambda - I_a^* - I_s^*)\frac{f_s(I_s^*)}{I_a^*}, \\
a_{22} &= -p(f_a(I_a^*)+f_s(I_s^*)) + p(\Lambda - I_a^* - I_s^*)f_s'(I_s^*) \; - \; (m + \sigma_s) \\
&= -p(f_a(I_a^*)+f_s(I_s^*)) + p(\Lambda - I_a^* - I_s^*)f_s'(I_s^*) - p(\Lambda - I_a^* - I_s^*)\frac{(f_a(I_a^*)+f_s(I_s^*))}{I_s^*} - \gamma\frac{I_a^*}{I_s^*} \\
&= -p(f_a(I_a^*)+f_s(I_s^*)) + p(\Lambda - I_a^* - I_s^*)\left(f_s'(I_s^*) - \frac{f_s(I_s^*)}{I_s^*}\right) - p(\Lambda - I_a^* - I_s^*)\frac{f_a(I_a^*)}{I_s^*} - \gamma\frac{I_a^*}{I_s^*} \\
&\leq -p(f_a(I_a^*)+f_s(I_s^*)) - p(\Lambda - I_a^* - I_s^*)\frac{f_a(I_a^*)}{I_s^*} - \gamma\frac{I_a^*}{I_s^*}, \\
a_{12} &= -(1-p)(f_a(I_a^*)+f_s(I_s^*)) + (1-p)(\Lambda - I_a^* - I_s^*)f_s'(I_s^*), \\
a_{21} &= -p(f_a(I_a^*)+f_s(I_s^*)) + p(\Lambda - I_a^* - I_s^*)f_a'(I_a^*) + \gamma.
\end{aligned}$$

The trace is given by

$$\text{Trace } (J^*) \leq -(f_a(I_a^*)+f_s(I_s^*)) - (1-p)(\Lambda - I_a^* - I_s^*)\frac{f_s(I_s^*)}{I_a^*} - p(\Lambda - I_a^* - I_s^*)\frac{f_a(I_a^*)}{I_s^*} - \gamma\frac{I_a^*}{I_s^*} < 0$$

and the determinant is given by

$$\begin{aligned}
\text{Det } (J^*) \;\geq\; & \Big(-(1-p)(f_a(I_a^*)+f_s(I_s^*)) - (1-p)(\Lambda - I_a^* - I_s^*)\frac{f_s(I_s^*)}{I_a^*}\Big)\times \\
& \Big(-p(f_a(I_a^*)+f_s(I_s^*)) - p(\Lambda - I_a^* - I_s^*)\frac{f_a(I_a^*)}{I_s^*} - \gamma\frac{I_a^*}{I_s^*}\Big) \\
& -\Big(-(1-p)(f_a(I_a^*)+f_s(I_s^*)) + (1-p)(\Lambda - I_a^* - I_s^*)f_s'(I_s^*)\Big)\times \\
& \Big(-p(f_a(I_a^*)+f_s(I_s^*)) + p(\Lambda - I_a^* - I_s^*)f_a'(I_a^*) + \gamma\Big) \\
= \;& p(1-p)(\Lambda - I_a^* - I_s^*)(f_a(I_a^*)+f_s(I_s^*))\Big(\frac{f_a(I_a^*)}{I_s^*} + \frac{f_a(I_s^*)}{I_a^*}\Big) \\
& + p(1-p)(\Lambda - I_a^* - I_s^*)^2\left(\frac{f_s(I_s^*)}{I_a^*}\frac{f_a(I_a^*)}{I_s^*} - f_a'(I_a^*)f_s'(I_s^*)\right) \\
& + \gamma(1-p)(f_a(I_a^*)+f_s(I_s^*))\left(\frac{I_a^*}{I_s^*}+1\right) + \gamma(1-p)(\Lambda - I_a^* - I_s^*)\left(\frac{f_s(I_s^*)}{I_s^*} - f_s'(I_s^*)\right) \\
& + p(1-p)(\Lambda - I_a^* - I_s^*)(f_a(I_a^*)+f_s(I_s^*))(f_a'(I_a^*)+f_s'(I_s^*)) > 0
\end{aligned}$$

By Lemma 1, the functions f_a and f_s satisfy $f_a'(I)I \leq f_a(I)$ and $f_s'(I)I \leq f_s(I)$, $\forall I \geq 0$ and since $(\Lambda - I_a^* - I_s^*) > 0$, then, all terms of $\text{Det}(J^*)$ are either positive or non-negative and therefore $\text{Det}(J^*) > 0$ and the equilibrium point E^* (which exists only if $\mathcal{R} > 1$) is LAS. □

3.2. Global analysis

Here, we discuss the global behaviour of the equilibrium points. For proving the global stability of the positive equilibrium points E_0 and E^*, we will exclude the existence of periodic solutions of system (3.2) by using generalized Bendixson-Dulac theorem. We first prove that the system (3.2) has no periodic orbit nor poly-cycle on Ω

Theorem 3. *System (3.2) has no periodic orbits nor poly-cycles on* Ω.

Proof. Consider a solution of system (3.2) on Ω. Let $\xi_1 = \ln(I_a)$ and $\xi_2 = \ln(I_s)$ then the transformation of (3.2) gives the following new system:

$$\begin{cases} \dot{\xi_1} &= h_1(\xi_1, \xi_2) := (1-p)(\Lambda - e^{\xi_1} - e^{\xi_2})(f_a(e^{\xi_1}) + f_s(e^{\xi_2}))e^{-\xi_1} - (\gamma + m + \sigma_a), \\ \dot{\xi_2} &= h_2(\xi_1, \xi_2) := p(\Lambda - e^{\xi_1} - e^{\xi_2})(f_a(e^{\xi_1}) + f_s(e^{\xi_2}))e^{-\xi_2}) + \gamma e^{\xi_1} e^{-\xi_2} - (m + \sigma_s). \end{cases} \tag{3.8}$$

We have

$$\begin{aligned} \frac{\partial h_1}{\partial \xi_1} + \frac{\partial h_2}{\partial \xi_2} &= -\Big(f_a(e^{\xi_1}) + f_s(e^{\xi_2})\Big) - \gamma e^{\xi_1} e^{-\xi_2} - (\Lambda - e^{\xi_1} - e^{\xi_2})\Big((1-p) f_s(e^{\xi_2}) e^{-\xi_1} + p f_a(e^{\xi_1}) e^{-\xi_2}\Big) \\ &\quad -(1-p)(\Lambda - e^{\xi_1} - e^{\xi_2})\Big(f_a(e^{\xi_1}) - f_a'(e^{\xi_1}) e^{\xi_1}\Big) e^{-\xi_1} - p(\Lambda - e^{\xi_1} - e^{\xi_2})\Big(f_s(e^{\xi_2}) - f_s'(e^{\xi_2}) e^{\xi_2}\Big) e^{-\xi_2} \\ &\quad < 0 \quad \forall\, \xi_1, \xi_2 \in \mathbb{R}. \end{aligned}$$

Thus using Dulac criterion [17, 18], system (3.8) has no periodic trajectory. Therefore system (3.2) has no periodic orbit inside $\otimes$. □

Theorem 4. *The solution of (3.2) converges asymptotically to :*

- E_0 *if* $\mathcal{R} < 1$.
- E^* *if* $\mathcal{R} > 1$.

Proof. We restrict the proof to the case where $\mathcal{R} > 1$. The other case can be done similarly. The system (3.2) admits two equilibrium points E_0 and E^*. E_0 is an unstable node, and only E^* is a stable node. We aim to prove that E^* is globally asymptotically stable. Let $I_a(0) > 0, I_s(0) > 0$ and ω the ω-limit set of $(I_a(0), I_s(0))$. ω is an invariant compact set and $\omega \subset \bar{\Omega}$. M can't be E_0 because E_0 is an unstable node and can't be a part of the ω-limit set of $(I_a(0), I_s(0))$. System (3.2) has no periodic orbit inside Ω. Using the Poincaré-Bendixon Theorem [17, 18], E^* is a globally asymptotically stable equilibrium point for system (3.2). □

4. Optimal control problem

The necessary conditions that an optimal pair must satisfy come from Pontryagin's Maximum Principle. Our goal is to minimize the number of infected individuals either asymptomatic or symptomatic while keeping those corresponding cost of the quarantine strategies low during the epidemic. Thus, we define a control function set as $V = \{v_1, v_2\}$ where $v_1(t)$ and $v_1(t)$ are the control variables for the quarantine strategies measuring the effort to reduce the contact between susceptible individuals and both kind of infected individuals (asymptomatic and symptomatic), respectively.

Therefore the model (2.1) is modified to the following new model:

$$\begin{cases} \dot{S} &= m\Lambda \ - \ S((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) \ + \ \sigma_a I_a \ + \ \sigma_s I_s \ - \ mS, \\ \dot{I}_a &= (1-p)S((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) \ - \ \gamma I_a \ - \ mI_a \ - \ \sigma_a I_a, \\ \dot{I}_s &= p\,S((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) + \gamma I_a \ - \ mI_s \ - \ \sigma_s I_s, \end{cases} \tag{4.1}$$

Several optimal strategies for epidemic models were proposed in several previous works [19–22]. In our case, we discussed an optimal strategy that reduces the contact between infected and uninfected individuals to optimise the number of infective individuals.

Recall that the set $\Omega = \{(S, I_a, I_s) \in \mathbb{R}^3_+ \ / \ S + I_a + I_s = \Lambda\}$ is a positively invariant compact set for (4.1).

Assume that f_a and f_s are globally Lipschitz with Lipschitz constants L_a and L_s where $\bar{f_a} = \sup_{I>0} f_a(I)$ and $\bar{f_s} = \sup_{I>0} f_s(I)$. Define the space

$$v_{ad} = \left\{(v_1, v_2)|\ v_1, v_2 \text{ Lebesque measurable}, 0 < \bar{v}_1 \le v_1 \le 1, 0 < \bar{v}_2 \le v_2 \le 1,\ 0 \le t \le T\right\}$$

where $\bar{v}_1$ and $\bar{v}_1$ are two non-negative constants.

Our goal is to control the number infected individuals and minimize the cost of the effort to reduce the contact between susceptible and infected individuals (v_1, v_2). The optimal control problem considered here is by minimizing an objective functional $J(v_1, v_2)$ as following

$$\min_{v_1, v_2 \in v_{ad}} J(v_1, v_2) = \min_{v_1, v_2 \in v_{ad}} \int_0^T \left(\frac{\alpha_1}{2} v_1^2(t) + \frac{\alpha_2}{2} v_2^2(t) + \beta_1 I_a(t) + \beta_2 I_s(t)\right) dt \ \text{ subject to (4.1).} \tag{4.2}$$

β_1 and β_2 are two constants.

Let $\varphi = (S, I_a, I_s)^t$, then we express (4.1) as

$$\dot{\varphi} = B\varphi + \psi(\varphi) = Z(\varphi) \tag{4.3}$$

where $B = \begin{pmatrix} -m & \sigma_a & \sigma_s \\ 0 & -(m+\sigma_a+\gamma) & 0 \\ 0 & \gamma & -(m+\sigma_s) \end{pmatrix}$ and

$$\psi(\varphi) = \begin{pmatrix} m\Lambda - S((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) \\ (1-p)S((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) \\ pS((1-v_1)f_a(I_a)+(1-v_2)f_s(I_s)) \end{pmatrix}.$$

Proposition 3. *$Z(\varphi)$ is a Lipschitz continuous function.*

Proof. First we shall prove that the function ψ is uniformly Lipschitz and continuous since

$$\begin{aligned}
|\psi(\varphi_1) - \psi(\varphi_2)| &= 2(1-v_1)\Big|f_a(I_{a1})S_1 - f_a(I_{a2})S_2\Big| + 2(1-v_2)\Big|f_s(I_{s1})S_1 - f_s(I_{s2})S_2\Big| \\
&= 2(1-v_1)\Big|S_1(f_a(I_{a1}) - f_a(I_{a2})) + f_a(I_{a2})(S_1 - S_2)\Big| \\
&\quad +2(1-v_2)\Big|S_1(f_s(I_{s1}) - f_s(I_{s2})) + f_s(I_{s2})(S_1 - S_2)\Big| \\
&\leq 2(1-\bar{v}_1)\Lambda\Big|f_a(I_{a1}) - f_a(I_{a2})\Big| + 2(1-\bar{v}_1)\bar{f}_a\Big|(S_1 - S_2)\Big| \\
&\quad +2(1-\bar{v}_2)\Lambda\Big|f_s(I_{s1}) - f_s(I_{s2})\Big| + 2(1-\bar{v}_2)\bar{f}_s\Big|(S_1 - S_2)\Big| \\
&\leq 2(1-\bar{v}_1)\Lambda L_a\Big|I_{a1} - I_{a2}\Big| + 2(1-\bar{v}_1)\bar{f}_a\Big|(S_1 - S_2)\Big| \\
&\quad +2(1-\bar{v}_2)\Lambda L_s\Big|I_{s1} - I_{s2}\Big| + 2(1-\bar{v}_2)\bar{f}_s\Big|(S_1 - S_2)\Big| \\
&\leq M|\varphi_1 - \varphi_2|
\end{aligned}$$

where $M = 2\max\Big(2(1-\bar{v}_1)\Lambda L_a, 2(1-\bar{v}_1)\bar{f}_a, 2(1-\bar{v}_2)\Lambda L_s, 2(1-\bar{v}_2)\bar{f}_s\Big)$.

$$|B\varphi_1 - B\varphi_2| \leq \|B\||\varphi_1 - \varphi_2| \tag{4.4}$$

where $\|.\|$ is the matrix norm. Then $|Z(\varphi_1) - Z(\varphi_2)| \leq H|\varphi_1 - \varphi_2|$ where the constant $H = \max(M, \|B\|)$. Therefore Z is Lipschitz continuous function. □

As the function ψ is uniformly Lipschitz and continuous then there exists a unique solution for the system (4.3). First, introduce the Lagrangian for the optimal problem (4.1) and (4.2)

$$L(S, I_a, I_s, v_1, v_2) = \frac{\alpha_1}{2}v_1^2 + \frac{\alpha_2}{2}v_2^2 + \beta_1 I_a + \beta_2 I_s. \tag{4.5}$$

We derive necessary conditions on the optimal control by applying Pontryagin's Maximum Principle [23]. Let the Hamiltonian H for the control problem (4.1) and (4.2):

$$\begin{aligned}
H(S, I_a, I_s, v_1, v_2, \lambda_1, \lambda_2, \lambda_3) &= \frac{\alpha_1}{2}v_1^2 + \frac{\alpha_2}{2}v_2^2 + \beta_1 I_a + \beta_2 I_s + \lambda_1\dot{S} + \lambda_2\dot{I}_a + \lambda_3\dot{I}_s \\
&= \frac{\alpha_1}{2}v_1^2 + \frac{\alpha_2}{2}v_2^2 + \beta_1 I_a + \beta_2 I_s \\
&\quad +\lambda_1\Big(m\Lambda - S((1-v_1)f_a(I_a) + (1-v_2)f_s(I_s)) + \sigma_a I_a + \sigma_s I_s - mS\Big) \\
&\quad +\lambda_2\Big((1-p)S((1-v_1)f_a(I_a) + (1-v_2)f_s(I_s)) - \gamma I_a - mI_a - \sigma_a I_a\Big) \\
&\quad +\lambda_3\Big(pS((1-v_1)f_a(I_a) + (1-v_2)f_s(I_s)) + \gamma I_a - mI_s - \sigma_s I_s\Big)
\end{aligned} \tag{4.6}$$

where λ_1, λ_2 and λ_3 are the adjoint variables satisfying the following adjoint equations

$$\begin{cases}
\dot{\lambda}_1 = -\dfrac{\partial H}{\partial S} = m\lambda_1 + \Big((1-v_1)f_a(I_a) + (1-v_2)f_s(I_s)\Big)\Big(\lambda_1 - (1-p)\lambda_2 - p\lambda_3\Big), \\
\dot{\lambda}_2 = -\dfrac{\partial H}{\partial I_a} = -\beta_1 - \sigma_a\lambda_1 + (\gamma + m + \sigma_a)\lambda_2 - \gamma\lambda_3 + S(1-v_1)f_a'(I_a)\Big(\lambda_1 - (1-p)\lambda_2 - p\lambda_3\Big), \\
\dot{\lambda}_3 = -\dfrac{\partial H}{\partial I_s} = -\beta_2 + S((1-v_2)f_s'(I_s)\Big(\lambda_1 - (1-p)\lambda_2 - p\lambda_3\Big) - \sigma_s\lambda_1 + \lambda_3(m + \sigma_s).
\end{cases} \tag{4.7}$$

Final conditions are given as follows: $\lambda_1(T) = 0, \lambda_2(T) = 0, \lambda_3(T) = 0$.

The Hamiltonian is minimized with respect to the control variables as following:

$$\min_{v_1,v_2,\lambda_1,\lambda_2,\lambda_3} H(S, I_a, I_s, v_1, v_2, \lambda_1, \lambda_2, \lambda_3, t). \tag{4.8}$$

The derivative of the Hamiltonian H with respect to v_1, and v_2 is given by

$$\frac{\partial H}{\partial v_1} = \alpha_1 v_1 + S f_a(I_a)(\lambda_1 - (1-p)\lambda_2 - p\lambda_3), \ \frac{\partial H}{\partial v_2} = \alpha_2 v_2 + S f_s(I_s)(\lambda_1 - (1-p)\lambda_2 - p\lambda_3). \tag{4.9}$$

The optimal control is obtained by resolving the necessary conditions: $\frac{\partial H}{\partial v_1} = 0$ and $\frac{\partial H}{\partial v_2} = 0$ on some non-trivial intervals. In this case, the controls are expressed as

$$v_1^*(t) = \frac{S f_a(I_a)(-\lambda_1 + (1-p)\lambda_2 + p\lambda_3)}{\alpha_1} \text{ and } v_2^*(t) = \frac{S f_s(I_s)(-\lambda_1 + (1-p)\lambda_2 + p\lambda_3)}{\alpha_2}$$

once

$$\bar{v}_1 \leq \frac{S f_a(I_a)(-\lambda_1 + (1-p)\lambda_2 + p\lambda_3)}{\alpha_1} \leq 1 \text{ and } \bar{v}_2 \leq \frac{S f_s(I_s)(-\lambda_1 + (1-p)\lambda_2 + p\lambda_3)}{\alpha_2} \leq 1.$$

5. Numerical simulations

For all numerical simulations, we used the Monod function as a non-linear incidence rates satisfying the assumption 1 $f_a(I) = \frac{\eta_a I}{\kappa_a + I}$ and $f_s(I) = \frac{\eta_s I}{\kappa_s + I}$. Here η_a, κ_a, η_s and κ_s are non-negative constants. The parameters used for the numerical simulations are given in Table 1. We begin by some numerical

Table 2. Used parameters for numerical simulations.

Parameter	κ_a	κ_s	σ_a	σ_s	m	γ	Λ	α_1	α_2	β_1	β_2
Value	2	1	0.4	0.5	1.5	0.1	8	1	1	1	1

results that confirm the stability of the equilibrium points of (3.2). In Figure 2, we give the results for the case where $\mathcal{R} < 1$. The numerical solution of the given model (3.2) approaches to the $DFE = (0, 0)$, which confirms that DFE is GAS when $\mathcal{R} < 1$.

In Figures 3 and 4, we give the results for the case where $\mathcal{R} > 1$. The numerical solution of given model (3.2) approaches asymptotically to the EE, which confirms that EE is GAS when $\mathcal{R} > 1$.

Sensitivity analysis can be helpful as to how the variability in the output of a mathematical model can be allocated to different sources of uncertainty in its input parameters [24,25]. Sensitivity analysis has several purposes; one is to determine the input parameters that most contribute to the system's dynamics. The other is to detect the impacts of each parameter on the other parameters and then determine the potential to simplify the model. In this study, the sensitivity study can be helpful as it will inform us how essential each parameter is to the transmission of the disease. We must discover the highest effect on the $\mathcal{R}$. Therefore, these input parameters will be critical targets for future intervention strategies. A fundamental approach expresses a relative change of a variable by a relative change of a parameter. Consequently, if the sensitivity index is positive, increasing the parameter value will cause

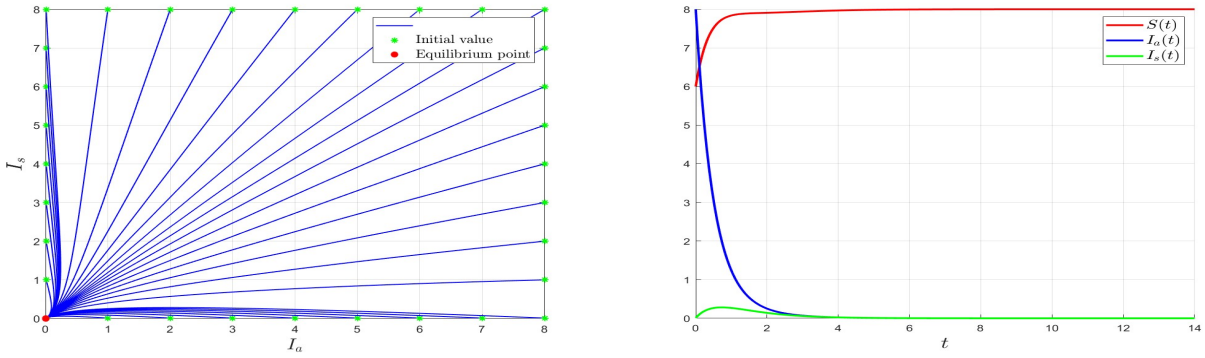

Figure 2. For $\eta_a = 0.1$ and $\eta_s = 0.2$ then $\mathcal{R} = 0.58 < 1$.

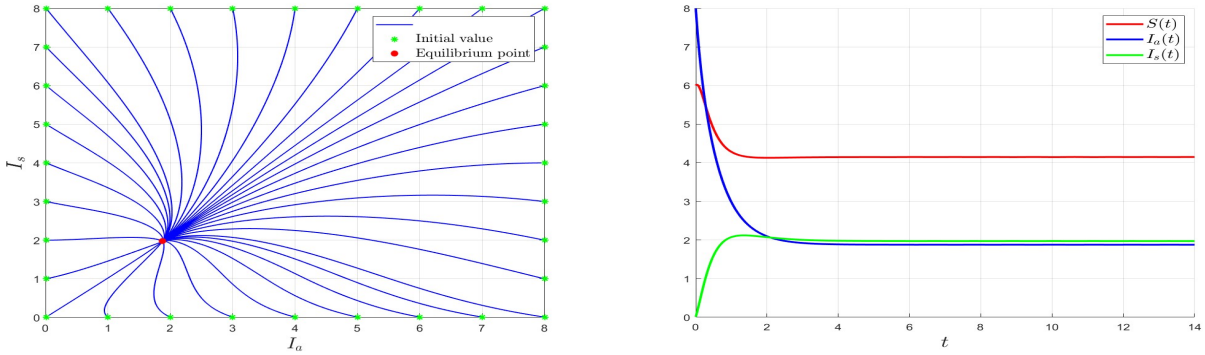

Figure 3. For $\eta_a = 1$ and $\eta_s = 2$ then $\mathcal{R} = 5.8 > 1$.

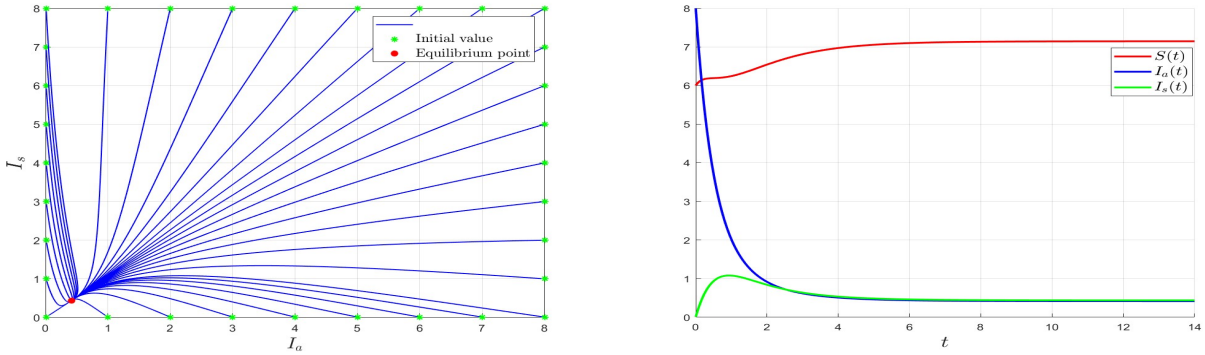

Figure 4. For $\eta_a = 1$ and $\eta_s = 0.2$ then $\mathcal{R} = 1.48 > 1$.

an increase in the $\mathcal{R}$ value. Further, if the result is negative, then the parameter value and the $\mathcal{R}$ are inversely proportional.

Figure 5 shows that Λ, p and γ are the only three parameters with a positive sign. Therefore, as the value of Λ, p and γ increase, the value of $\mathcal{R}$ increases. Furthermore, since the remaining parameters increases, the value of $\mathcal{R}$ decreases. Figure 5 shows the behaviour of the $\mathcal{R}$ with respect to the model parameters.

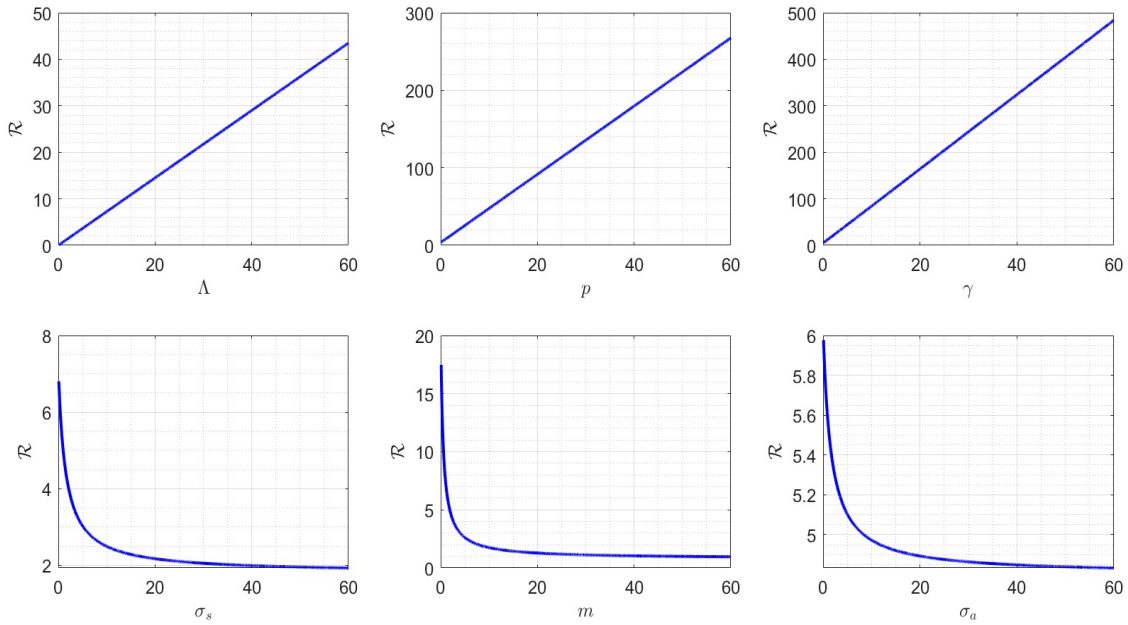

Figure 5. Sensitivity of the reproduction number $\mathcal{R}$ with respect to the model parameters.

The numerical results of the control problem were obtained using the an association between the Gauss-Seidel-like implicit finite-difference scheme and the first-order backward-difference.
We used the same parameters for systems (4.1) and (4.2) as for the direct problem (3.2) with $\eta_a = 1$ and $\eta_s = 2$ (left) and with $\eta_a = 1$ and $\eta_s = 0.2$ (where EE is GAS) with variables v_1 and v_2 such that the initial condition $v_1(0) = v_2(0) = 0.5$. We plot in Figure 6 the behaviours $S(t), I_a(t)$ and $I_s(t)$ with respect to time.

As seen in Figure 2, susceptible compartment increases about 21%, however, the infected

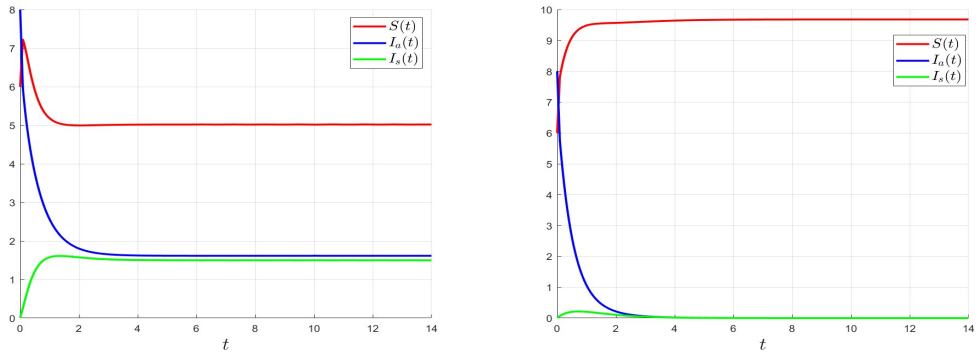

Figure 6. Behaviours for $\eta_a = 1$ and $\eta_s = 2$ (left) and for $\eta_a = 1$ and $\eta_s = 0.2$ (right).

compartment decreases about 14% for the asymptomatic infected compartment and 14% for the asymptomatic infected compartment of their values at steady state in Figure 2. Note that there is no

considerable influence of the values of $\alpha_1, \alpha_2, \beta_1$ and β_2 on the final values of susceptible and both infected compartments.

6. Conclusions

In the present paper, a generalized "SIS" model with a non-linear incidence rate including asymptomatic and symptomatic infection was presented and analyzed. The basic reproduction number ($\mathcal{R}$) was calculated for the proposed system using the next generation matrix method. The system admits at most two equilibria: the disease-free equilibrium (E_0) and the endemic equilibrium (E^*). Local and global stability was carried. If $\mathcal{R} < 1$, the disease-free equilibrium E_0 is locally and globally asymptotically stable; if $\mathcal{R} > 1$, the endemic equilibrium E^* is locally and globally asymptotically stable. Furthermore, we find several reasonable optimal control strategies to the prevention and control the disease. Finally, numerical simulations are presented to verify the above theoretical results are given. It is concluded that asymptomatic infections have a greater contribution to the diseases spread even in the case where the asymptomatic infections are less infectious.

One of the compartmental models' limitations is the assumption of a homogeneous population. However, variations in individuals in epidemiological characteristics are more realistic to consider. Further, the influences of time delay and intrinsic fluctuations are considered limitations to the compartmental models.

Competing interests

The author declares no conflict of interest.

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Appendix

Used numerical scheme (control problem)

Subdividing the interval $[0,T]$ as the following $[0,T] = \bigcup_{n=0}^{N-1}[t_n, t_{n+1}]$ where $t_n = ndt$ and $dt = T/N$. Let S^n, I_a^n, I_s^n, λ_1^n, λ_2^n, λ_3^n, v_1^n and v_2^n approximate $S(t), I_a(t), I_s(t), \lambda_1(t), \lambda_2(t), \lambda_3(t)$, $v_1(t)$ and $v_2(t)$, respectively at the time t_n. $S^0, I_a^0, I_s^0, \lambda_1^0, \lambda_2^0, \lambda_3^0, v_1^0$ and v_2^0 be their values at initial time ($t = 0$). S^N, I_a^N, I_s^N, $\lambda_1^N, \lambda_2^N, \lambda_3^N, v_1^N$ and v_2^N be the values at $t = T$. Then, we use the following scheme [6, 8, 21, 22]

$$
\begin{cases}
\dfrac{S^{n+1}-S^n}{\delta t} = m\Lambda - S^{n+1}((1-v_1^n)f_a(I_a^n) + (1-v_2^n)f_s(I_s^n)) + \sigma_a I_a^n + \sigma_s I_s^n - mS^{n+1},\\
\dfrac{I_a^{n+1}-I_a^n}{\delta t} = (1-p)S^{n+1}((1-v_1^n)f_a(I_a^n) + (1-v_2^n)f_s(I_s^n)) - \gamma I_a^{n+1} - mI_a^{n+1} - \sigma_a I_a^{n+1},\\
\dfrac{I_s^{n+1}-I_s^n}{\delta t} = p\ S^{n+1}((1-v_1^n)f_a(I_a^{n+1}) + (1-v_2^n)f_s(I_s^n)) + \gamma I_a^{n+1} \ - \ mI_s^{n+1} \ - \ \sigma_s I_s^{n+1},\\
\dfrac{\lambda_1^{N-n}-\lambda_1^{N-n-1}}{\delta t} = m\lambda_1^{N-n-1} + \Big((1-v_1^n)f_a(I_a^{n+1}) + (1-v_2^n)f_s(I_s^{n+1})\Big)\Big(\lambda_1^{N-n-1} - (1-p)\lambda_2^{N-n} - p\lambda_3^{N-n}\Big),\\
\dfrac{\lambda_2^{N-n}-\lambda_2^{N-n-1}}{\delta t} = -\beta_1 - \sigma_a\lambda_1^{N-n-1} + (\gamma + m + \sigma_a)\lambda_2^{N-n-1} - \gamma\lambda_3^{N-n}\\
\qquad\qquad +S^{n+1}(1-v_1^n)f_a'(I_a^{n+1})\Big(\lambda_1^{N-n-1} - (1-p)\lambda_2^{N-n-1} - p\lambda_3^{N-n}\Big),\\
\dfrac{\lambda_3^{N-n}-\lambda_3^{N-n-1}}{\delta t} = -\beta_2 + S^{n+1}(1-v_2^n)f_s'(I_s^{n+1})\Big(\lambda_1^{N-n-1} - (1-p)\lambda_2^{N-n-1} - p\lambda_3^{N-n-1}\Big) - \sigma_s\lambda_1^{N-n-1}\\
\qquad\qquad +\lambda_3^{N-n-1}(m+\sigma_s).
\end{cases}
$$

Therefore the following algorithm will be applied.

$S^0 \leftarrow S_0, I_a^0 \leftarrow I_{a0}, I_s^0 \leftarrow I_{s0}, \lambda_1^N \leftarrow 0, \lambda_2^N \leftarrow 0, \lambda_3^N \leftarrow 0,$
for $n = 0$ to $N-1$ **do**

$$
\begin{aligned}
S^{n+1} &\leftarrow \frac{S^n + \delta t(m\Lambda + \sigma_a I_a^n + \sigma_s I_s^n)}{1 + \delta t(m + (1-v_1^n) f_a(I_a^n) + (1-v_2^n) f_s(I_s^n))},\\
I_a^{n+1} &\leftarrow \frac{I_a^n + \delta t(1-p) S^{n+1}((1-v_1^n) f_a(I_a^n) + (1-v_2^n) f_s(I_s^n))}{1 + \delta t(\gamma + m + \sigma_a)},\\
I_s^{n+1} &\leftarrow \frac{I_s^n + \delta t(p\, S^{n+1}((1-v_1^n) f_a(I_a^{n+1}) + (1-v_2^n) f_s(I_s^n)) + \gamma I_a^{n+1})}{1 + \delta t(m + \sigma_s)},\\
\lambda_1^{N-n-1} &\leftarrow \frac{\lambda_1^{N-n} + \delta t\Big((1-v_1^n) f_a(I_a^{n+1}) + (1-v_2^n) f_s(I_s^{n+1})\Big)\Big((1-p)\lambda_2^{N-n} + p\lambda_3^{N-n}\Big)}{1 + \delta t\Big(m + (1-v_1^n) f_a(I_a^{n+1}) + (1-v_2^n) f_s(I_s^{n+1})\Big)},\\
\lambda_2^{N-n-1} &\leftarrow \frac{\lambda_2^{N-n} + \delta t(\beta_1 + \sigma_a \lambda_1^{N-n-1} + \gamma\lambda_3^{N-n} - S^{n+1}(1-v_1^n) f_a'(I_a^{n+1})\Big(\lambda_1^{N-n-1} - p\lambda_3^{N-n}\Big)}{1 + \delta t(\gamma + m + \sigma_a - (1-p) S^{n+1}(1-v_1^n) f_a'(I_a^{n+1}))},\\
\lambda_3^{N-n-1} &\leftarrow \frac{\lambda_3^{N-n} + \delta t(\beta_2 - S^{n+1}(1-v_2^n) f_s'(I_s^{n+1})\Big(\lambda_1^{N-n-1} - (1-p)\lambda_2^{N-n-1}\Big) + \sigma_s \lambda_1^{N-n-1}}{1 + \delta t(m + \sigma_s - pS^{n+1}(1-v_2^n) f_s'(I_s^{n+1}))},\\
v_1^{n+1} &\leftarrow \max(\min(\frac{S^{n+1} f_a(I_a^{n+1})(-\lambda_1^{N-n-1} + (1-p)\lambda_2^{N-n-1} + p\lambda_3^{N-n-1})}{\alpha_1}, 1), 0),\\
v_2^{n+1} &\leftarrow \max(\min(\frac{S^{n+1} f_s(I_s^{n+1})(-\lambda_1^{N-n-1} + (1-p)\lambda_2^{N-n-1} + p\lambda_3^{N-n-1})}{\alpha_2}, 1), 0).
\end{aligned}
$$

end

Algorithm 1: Optimal control resolution