Available online at http://scik.org
J. Math. Comput. Sci. 2025, 15:15
https://doi.org/10.28919/jmcs/9533
ISSN: 1927-5307

GLOBAL STABILITY AND OPTIMAL CONTROL OF SIRS MODEL WITH MEDIA COVERAGE: ANALYSIS OF INFECTED AND SUSCEPTIBLE POPULATION

AMERA H. ALMUSHARRF*

Jubail Industrial College, Jubail Industrial City, Jubail 31961, The Kingdom of Saudi Arabia

Abstract. In this paper a system of SIRS epidemic model with media coverage is considered. We perform a study of global stability analysis, and sensitivity analysis of the basic reproduction number R_0. The system is globally asymptotically stable about the endemic equilibrium if the basic reproduction number $R_0 > 1$. The geometric approach is used to prove the global stability of the endemic equilibrium if the basic reproduction number $R_0 > 1$. R_0 depends on a set of positive parameters. The sensitivity indices of those parameters are calculated by using the normalized sensitivity formula and can be classified into two classes: one class has a positive correlation with R_0, and the other class has a negative correlation with R_0. Furthermore, Optimal control is applied to explore the possible control strategies to prevent disease spread in the community. We extend the proposed SIRS model to include three control variables namely educational campaign, vaccination, and treatment care. Using Pontryagin's maximal principle, we established the necessary conditions for the existence of optimal control. We use the fourth-order Runge Kutta forward-backward sweep approach to simulate the optimality system in order to demonstrate the impact of various combinations of controls on the spread of disease. A cost-effectiveness study is carried out to inform the public about the best cost-effective technique among several control combinations. The results suggest that, the preventative tactics through educational campaigns is the most cost-effective.

Keywords: SIRS model; global stability; geometric approach; optimal control.

2020 AMS Subject Classification: 92D30, 49K15, 34A34.

*Corresponding author

E-mail address: ameraalmusharrf@gmail.com

Received August 04, 2025

1. INTRODUCTION

An epidemic is defined as an unusually large, short-term disease outbreak. Various factors influence a disease's spread from person to person. These include the infectious agent itself, its mode of transmission, infectious period, and its susceptibility and resistance to treatments and vaccines. Additionally, factors within the population contribute. These include social, demographic, cultural, geographic, and economic factors.

In the early 20th century, mathematical modeling was introduced into the field of epidemiology by scientists such as Anderson Gray McKendrick and Janet-Leigh Claypon. Since then, mathematical modeling has increasingly played an intrinsic part in managing outbreaks and epidemics and informing public health decisions[20].

The first mathematical model of infectious disease transmission was constructed by Bernoulli in 17601 to determine the impact of variolation, a crude form of smallpox vaccination, on life-tables used for actuarial purposes[21]. Various models have been used to analyze different properties of diseases spreading [1]-[19]. A general SIRS epidemic model can be formulated as

$$\begin{cases} \dfrac{dS}{dt} = r - dS - f(I)SI + \delta R, \\ \dfrac{dI}{dt} = f(I)SI - (d+\gamma+\alpha)I, \\ \dfrac{dR}{dt} = \gamma I - (d+\delta)R, \end{cases} \tag{1.1}$$

where S, I, and R denote the susceptible, the infected, and the recovered populations, respectively. We assume all the parameters are positive, and

- r is the recruitment rate of the susceptible population;
- γ is the natural recovery rate infective individuals;
- d is the natural death rate and α is the disease-induced death rate ;
- δ is the the rate at which recovered individuals lose immunity.

The transmission of the infection is governed by the incidence rate $f(I)S$, $f(I)S$ is the infection force. $f(I)S$ has been considered to play a key role in ensuring that the models indeed give reasonable qualitative description of the transmission dynamics of the diseases. Some factors, such as media coverage, density of population, and life style, may affect the incidence rate

directly or indirectly. A number of mathematical models have been formulated to describe the impact of media coverage on the transmission dynamics of infectious diseases [13, 5, 5, 14, 15]. The media coverage in [5] is described as

$$f(I) = \beta_1 - \beta_2 \frac{I}{m+I} \tag{1.2}$$

where β_1 is the contact rate before media alert; the terms $\beta_2 \frac{I}{m+I}$ measure the effect of reduction of the contact rate when infectious individuals are reported on the media. The reduced value of the transmission rate approaches its maximum at β_2 when the reported infective number arrives at m. Since the coverage report cannot prevent disease from spreading completely we have $\beta_1 \geq \beta_2$. The half-saturation constant $m > 0$ reflects the impact of media coverage on the contact transmission. The function $\frac{I}{m+I}$is a continuous bounded function which takes into account disease saturation or psychological effect[14].

In this paper, we study the Model 1.1 with the media coverage function suggested in Equation 1.2. The paper is organized as follows. In Section 3, we carry out the study of global stability of the proposed model about the epidemic equilibrium point. In Section 4, we analyze the sensitivity Analysis of reproduction number R_0. In Sections 5,6,7, we explore the optimal control problem of the suggested model. Finally, in section 8, we calculate the Cost-Effectiveness of the introduced control strategists.

2. Mathematical Model

Based on the general SIRS model with the incidence rate, the model consist of the following differential equations:

$$\begin{cases} \frac{dS}{dt} = r - dS - \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) SI + \delta R, \\ \frac{dI}{dt} = \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) SI - (d+\gamma+\alpha) I, \\ \frac{dR}{dt} = \gamma I - (d+\delta) R, \end{cases} \tag{2.1}$$

If we set the initial condition as $S(0) \geq 0, I(0) \geq 0$ and $R(0) \geq 0.$, it is clear that the solutions to model 2.1 are nonnegative for $t > 0$.

Let $N = S+I+R$ then $\frac{dN}{dt} = r - dN - \alpha I \leq r - dN$, which follows that $N \leq \frac{r}{d}$. Therefore, the region

$$\Omega = \{(S,I,R) \in \mathbb{R}^3 | 0 < S+I+R \leq \frac{r}{d}, S \geq 0, I \geq 0, R \geq 0\}$$

is positive invariant domain of the system 2.1. Based on the next-generation matrix approach [9], the article [5] showed that the basic reproduction number R_0 as follows:

$$R_0 = \frac{r\beta_1}{d(d+\alpha+\gamma)}$$

R_0 measures the average number of secondary infections that occur when one infective is introduced into a completely host population [9, 11, 6]. The following theorem summarizes the dynamic for the system 2.1.

Theorem 2.1. [5] *Consider the model 2.1 with all positive parameters. If $R_0 < 1$, the model 2.1 has a unique disease-free equilibrium $E_0(S_0, I_0, R_0)$ and is globally stable; when $R_0 > 1$ the disease-free equilibrium becomes unstable and there is a unique positive endemic equilibrium $E^*(S^*, I^*, R^*)$ which is locally asymptotically stable.*

3. Global Stability of Endemic Equilibrium

The previous Theorem2.1 which was proven in [5] shows that the system 2.1 has a disease-free equilibrium E_0 which is globally stable when $R_0 < 1$, and the existence of a unique positive endemic equilibrium E^* when $R_0 > 1$ which is locally stable. The method of Lyapunov functions is most commonly used (see [22],[23]); its application is often hindered by the fact that in many cases global Lyapunov functions are difficult to construct and there is practically no general approach to the construction of such functions. An alternative approach to the global-stability problem has emerged from a series of papers on higher-dimensional generalizations of the criteria of Bendixson and Dulac for planar systems and on so-called autonomous convergence theorems [12].

In this section we will focus on the global stability of the endemic equilibrium if $R_0 > 1$. We follow the geometric method present in [6, 12, 8] to prove the global stability of the endemic equilibrium for the system 2.1. We briefly describe the geometric approach based on the second

additive compound matrix, developed by Li and Muldowney. For 3×3 matrix $A = [a_{ij}]$, the second additive compound matrix is defined as

$$A^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}.$$

Now consider the autonomous dynamical system

$$\frac{dX}{dt} = F(X), \tag{3.1}$$

where $F : D \to \mathbb{R}^n$ is C^1 function and where $D \subset \mathbb{R}^n$ is simply connected open set. Let $X(t, X_0)$ denote the solution of Equation 3.1 with the initial condition $X(0) = X_0$. We assume:

(H1) There exists a compact absorbing set $K \subset D$;

(H2) The system 3.1 has a unique equilibrium point X^* in D.

Then X^* is globally asymptotically stable if 3.1 satisfies (H1) and (H2) and a Bendixon criterion that is robust under C^1 of F. For more details, see [8]. This criterion is obtained as follows: Let $X \to P(X)$ be a $\binom{n}{a} \times \binom{n}{a}$ matrix -valued C^1 function in D. Set

$$Q = P_F P^{-1} + P J^{[2]} P^{-1}, \tag{3.2}$$

where P_F is the derivative of P (entry-wise) along the direction of F and $J^{[2]}$ is the second compound matrix of the Jacobian $J(X) = DF(X)$. Let $m(Q)$ be the Lozinskii measure with respect to a matrix norm [6], i.e.

$$m(Q) = \lim_{h \to 0^+} \frac{|I + hQ| - 1}{h}.$$

Define a quantity $\bar{q_2}$ as

$$\bar{q_2} = \lim_{t \to \infty} \sup \sup_{X_0 \in K} \frac{1}{t} \int_0^t m(Q(X(s, X_0)))\, ds.$$

The Bendixon criterion is given by

$$\bar{q_2} < 0.$$

In summary we have the following lemma.

Lemma 3.1. *Assume that D is simply connected and the assumptions (H1) and (H2) hold. Then the unique equilibrium X^* is globally asymptotically stable in D if $\bar{q}_2 < 0$.*

Next, to prove the global stability of the endemic equilibrium point E^*, we prove first that the system 2.1 is uniformly persistent.

Theorem 3.2. *If $R_0 > 1$, the system 2.1 is uniformly persistent, and the endemic equilibrium E^* is globally asymptotically stable in the interior of Ω.*

Proof. Theorem 2.1 shows that when $R_0 > 1$ there exists a unique positive endemic equilibrium E^* which is locally asympotocally stable. Therefore, the condition (H2) holds. When $R_0 > 1$, from Theorem 2.1 the disease free equilibrium $E_0(\frac{r}{d}, 0, 0)$ is unstable. Since E_0 is on the boundary of the domain Ω. This implies the system 2.1 is uniformly persistence. The uniform persistence with the boundedness of Ω imply that the system has a compact absorbing subset K of Ω [5, 6, 7]. Thus, the condition (H1) and (H2) hold. The Jacobian matrix J for the system 2.1 is given by

$$J = \begin{bmatrix} -d - f(I)I & -Sf(I) - SI\dfrac{df}{dI} & \delta \\ f(I)I & Sf(I) + SI\dfrac{df}{dI} - (d+\gamma+\alpha) & 0 \\ 0 & \gamma & -(d+\delta) \end{bmatrix}$$

and

$$J^{[2]} = \begin{bmatrix} \Delta - f(I)I & 0 & -\delta \\ \gamma & -(2d+\delta) - f(I)I & -Sf(I) - SI\dfrac{df}{dI} \\ 0 & f(I)I & \Delta + \delta \end{bmatrix},$$

where $\Delta = -(2d+\gamma+\alpha) + Sf(I) + SI\dfrac{df}{dI}$.

We set the matrix function P by

$$P(S,I,B) = diag\left\{1, \frac{I}{R}, \frac{I}{R}\right\}.$$

Then,

$$P_F P^{-1} = diag\left\{0, \frac{I'}{I} - \frac{R'}{R}, \frac{I'}{I} - \frac{R'}{R}\right\}$$

and $PJ^{[2]}P^{-1} =$

$$\begin{bmatrix} \Delta - f(I)I & 0 & -\frac{R}{I}\delta \\ \frac{I}{R}\gamma & -(2d+\delta) - f(I)I & -Sf(I) - SI\frac{df}{dI} \\ 0 & f(I)I & \Delta + \delta \end{bmatrix}.$$

The matrix $P_F P^{-1} + PJ^{[2]}P^{-1}$ defined in Equation 3.2 can be written in block form:

$$Q = \begin{bmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{bmatrix},$$

with

$$Q_{11} = -(2d+\gamma+\alpha) + f(I)(S-I) + SI\frac{df}{dI},$$

$$Q_{12} = \begin{bmatrix} 0, & -\frac{R}{I}\delta \end{bmatrix}, \quad Q_{21} = \begin{bmatrix} \frac{I}{R}\gamma \\ 0 \end{bmatrix},$$

and

$$Q_{22} = \begin{bmatrix} -(2d+\delta) - f(I)I + \frac{I'}{I} - \frac{R'}{R} & -Sf(I) - SI\frac{df}{dI} \\ f(I)I & \Delta + \delta + \frac{I'}{I} - \frac{R'}{R} \end{bmatrix}.$$

Now we define a norm in $\mathbb{R}^3$ as

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

For any vector $(u,v,w) \in \mathbb{R}^3$. Let m denote the Lozinskii measure with respect to this norm. We can then obtain

$$m(Q) \leq \sup\{g_1, g_2\}, \tag{3.3}$$

with

$$g_1 = m_1(Q_{11}) + |Q_{12}|,$$

$$g_2 = |Q_{21}| + m_1(Q_{22}),$$

where $|Q_{12}|$ and $|Q_{12}|$ are matrix norms induced by the L_1 vector norm, and m_1 denotes the Lozinskii measure with respect to the L_1 norm.

Now,

$$\begin{aligned} g_1 &= m_1(Q_{11}) + |Q_{12}| \\ &= \Delta - f(I)(I) + \frac{R}{I}\delta \\ &= -(2d+\gamma+\alpha) + f(I)(S-I) + SI\frac{df}{dI} + \frac{R}{I}\delta \end{aligned}$$

Based on the the second equation of system 2.1, we have

$$\frac{I'}{I} = f(I)S - (d+\gamma+\alpha)$$

Since the system 2.1 is uniformly persistent, then there exists $c > 0$ such that every solution $(S(t), I(t), R(t))$ of 2.1 with $(S(0), I(0), R(0))$ in the interior of Ω satisfies

$$\liminf_{t\to\infty} |(S(t), I(t), R(t))| \geq c$$

which implies for $t > T$

$$S(t) \geq c, \quad I(t) \geq c, \quad \text{and } R(t) \geq c$$

and since $R < N < \frac{d}{r}$, then $\frac{R}{I}\delta \leq \varepsilon$, where $\varepsilon = \frac{d}{rc}\delta$.
Set $\bar{\varepsilon} = \frac{rc}{2} < 1$. Then for $t > T$ and $\delta < \bar{\varepsilon}$

$$-d + \frac{R}{I}\delta \leq 0$$

Therefore,

$$\begin{aligned} g_1 &= \frac{I'}{I} - d - f(I)I + SIf' + \frac{R}{I}\delta \\ &\leq \frac{I'}{I} - (d-\varepsilon) \end{aligned}$$

Meanwhile,

$$\begin{aligned} g_2 &= -(2d+\delta) + \frac{I'}{I} - \frac{R'}{R} + 2\sup\left\{Sf(I) + SI\frac{df}{dI} - (\gamma+\alpha), 0\right\} \\ &\leq \frac{I'}{I} - d \\ &\leq \frac{I'}{I} - (d-\varepsilon) \end{aligned}$$

provided that

$$\max_{(S,I,R)\in\Omega}\left[Sf(I)+SI\frac{df}{dI}\right]\leq\gamma+\alpha$$

and from the third equations of the system 2.1 we have

$$\frac{R'}{R}=\gamma\frac{I}{R}-(d+\delta)$$

Therefore,

$$m(Q)\leq\frac{I'}{I}-(d-\varepsilon)$$

Since very solution $(S(t),I(t),R(t))$ of the system 2.1 with$(S(0),I(0),R(0))\in K$, where K is the compact absorbing set., there exists $t>T$ such that when $\frac{1}{t}[\ln(t)-\ln(0)]<(d-\varepsilon)/2$, consequently,

$$\frac{1}{t}\int_0^t m(Q)\,dt\leq\int_0^t\frac{I'}{I}-(d-\varepsilon)\,dt=\frac{\ln(t)-\ln(0)}{t}-(d-\varepsilon)<-\frac{(d-\varepsilon)}{2}$$

which implies $\bar{q_2}\leq-\frac{d-\varepsilon}{2}<0$, completing the proof of Theorem 2.1 □

4. Sensitivity Analysis of the Basic Reproduction Number R_0

It is a technique used to study the impact of a parameter on the model. For instance, if we increase a parameter would that result in increasing the dependent variable or not. The sensitivity index of each model parameter, which is connected with the basic reproduction number R_0 is calculated in this section. This index indicates the relative importance of each parameter in the model that depicts disease transmission. A parameter with large impact on R_0 imply that it has significant impact on the endemicity. Analyzing the impact of each parameter on the disease's epidemic, would allow us to have a better control on its spread. To derive the sensitivity index parameter of the basic reproduction number, same approach which was found in [10] is implemented. The sensitivity index of each parameter which is related to R_0 is calculated using the definition 4.1 as follows.

Definition 4.1. *(see* [10, 23]*) The normalized sensitivity index is calculated using the normalized sensitivity index of the variable R_0 , which is differentiable on the parameter p:*

$$C_p^{R_0}=\frac{\partial R_0}{\partial p}\frac{p}{R_0}.$$

Then, the sensitivity index of $R_0 = \dfrac{r\beta_1}{d(d+\alpha+\gamma)}$ to the parameter β_1, is given by

$$C_{\beta_1}^{R_0} = \frac{\partial R_0}{\partial \beta_1}\frac{\beta_1}{R_0} = 1.$$

TABLE 1. Sensitivity Indices of Parameters to R_0

Parameter	Sensitivity Index
r	1
β_1	0.002
d	-1.11765
α	-0.588235
γ	-0.294118

We use the following set values: $r = 5, d = 0.02, \delta = 0.01, \alpha = 0.1, \gamma = 0.05, \beta_1 = 0.002$, *and* $\beta_2 = 0.0018$[14]. The sensitivity index of each parameter is provided in Table 1. Both parameters β_1 and r have positive sensitivity indices, while the parameters d, α and γ have negative sensitivity indices. A parameter with a positive sensitivity index indicates that increasing (or decreasing) the value of that parameter while maintaining the value of the other parameters will lead to increases (or decreases) in the basic reproduction number. However, A parameter with negative sensitivity index indicate that increasing (or reducing) the value of the parameter while the values of the other parameters remain constant will result in decreases (or rises) in the basic reproduction number.

5. OPTIMAL CONTROL PROBLEM

In this section,we formulate the optimal control problem corresponding to Model 2.1 which minimizes both the infected and susceptible populations with minimum cost. To achieve this, we modify Model 2.1 by incorporating the triple of the control functions $u_1(t)$, $u_2(t)$ and $u_3(t)$, where u_1 represents the preventive strategies by by educational campaign, u_2 represents the control functions associated with vaccination which given to the susceptible population and u_3 denotes the control function targeted at reducing the infected population by treatment care. Consequently, the optimal control SIRS model with the triple time-dependent functions $u_1(t)$,

$u_2(t)$ and $u_3(t)$ is given by

$$\begin{cases} \dfrac{dS}{dt} = r - dS - (1-u_1)\left(\beta_1 - \beta_2 \dfrac{I}{m+I}\right)SI + \delta R - u_2 S, \\ \dfrac{dI}{dt} = (1-u_1)\left(\beta_1 - \beta_2 \dfrac{I}{m+I}\right)SI - (d+\gamma+\alpha)I - u_3 I, \\ \dfrac{dR}{dt} = \gamma I + u_2 S + u_3 I - (d+\delta)R. \end{cases} \tag{5.1}$$

Now, we define the cost functional as

$$J(u_1(t), u_2(t), u_3(t)) = \int_0^T [\frac{1}{2} w_1 u_1^2 + \frac{1}{2} w_2 u_2^2 + \frac{1}{2} w_3 u_3^2 + w_4 I]\, dt. \tag{5.2}$$

where T is the final time for control implementation, for $t \in [0,T]$. The weight constants w_1, w_2 *and* w_3 represent the cost associated with the preventive strategies by educational campaign,vaccination, and treatment. Moreover, the constant weight w_4 represents the cost associated for minimizing $I(t)$. For the sake of simplicity, we write u_i instead of $u_i(t)$ where $i = 1,2,3$. Therefore, the triple of optimal controls are sought such that

$$J(u^*) = min\{J(u_1(t), u_2(t), u_3(t)) : u_1, u_2, u_3 \in \mathbb{U}\}$$

where $\mathbb{U}$ is the non empty control set given as $\mathbb{U} = \{(u_1, u_2, u_3) : 0 \le u_1, u_2, u_3 \le 1, t \in [0,T]\}$. Following the existence results by [16, 17], the optimal control triple $u^* = (u_1^*, u_2^*, u_3^*)$ exists.

6. Necessary Conditions for the Control

The Pontryagin's Maximum Principle [16, 17, 18] gives the necessary conditions for the optimal control triple u^*. The Hamiltonian is defined as follows:

$$\begin{aligned} \mathscr{H}(S,I,R,u_1,u_2,u_3,\lambda_1,\lambda_2,\lambda_3) &= \frac{1}{2}(w_1 u_1^2 + w_2 u_2^2 + w_3 u_3^2) + w_4 I + \lambda_1 S' + \lambda_2 I' + \lambda_3 R \\ &= \frac{1}{2}(w_1 u_1^2 + w_2 u_2^2 + w_3 u_3^2) + w_4 I \\ &+ \lambda_1 \left(r - dS - (1-u_1)\left(\beta_1 - \beta_2 \frac{I}{m+I}\right)SI + \delta R - u_2 S \right) \\ &+ \lambda_2 \left[\left((1-u_1)\left(\beta_1 - \beta_2 \frac{I}{m+I}\right)\right)SI - (d+\gamma+\alpha)I - u_3 I \right] \\ &+ \lambda_3 \left(\gamma I + u_2 S + u_3 I - (d+\delta)R \right), \end{aligned}$$

where $\lambda = \lambda_i$ for $i = 1,2,3$ are adjoint functions associated with the state variables of the optimal control Model 5.1. By Pontryagin's maximum principle,the costate equations are given as follows:

$$\frac{d\lambda_1}{dt} = -\frac{\partial \mathscr{H}}{dS}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial \mathscr{H}}{dI} \text{ and } \frac{d\lambda_3}{dt} = -\frac{\partial \mathscr{H}}{dR},$$

with the following transversality conditions $\lambda_i(T) = 0$,for all $i = 1,2$. Therefore,

$$\begin{aligned}
\frac{d\lambda_1}{dt} &= \lambda_1(d + (1-u_1)f(I)I + u_2) - \lambda_2(1-u_1)f(I)I - \lambda_3 u_2,\\
\frac{d\lambda_2}{dt} &= -w_4 + \lambda_1\left[(1-u_1)\left(f(I)S + SI\frac{df(I)}{dI}\right)\right]\\
&\quad - \lambda_2\left[(1-u_1)\left((f(I)S + SI\frac{df(I)}{dI}\right) - (d+\gamma+\alpha) - u_3)\right] - \lambda_3(\gamma+u_3),\\
\frac{d\lambda_3}{dt} &= -\lambda_1\delta + \lambda_3(d+\delta).
\end{aligned}$$

By using the condition for optimality, we have

$$\frac{\partial \mathscr{H}}{\partial u_i} = 0 \text{ at } u_i = u_i^*, \quad \text{for } i = 1,2,3.$$

Therefore,we obtain

$$u_1^* = \frac{(\lambda_2 - \lambda_1)f(I)SI}{w_1}, u_2^* = \frac{(\lambda_1 - \lambda_3)S}{w_2}, \text{ and } u_3^* = \frac{(\lambda_2 - \lambda_3)I}{w_3}.$$

The optimal control triple (u_1^*, u_2^*, u_3^*) is is subject to the admissible conditions:

$$\begin{aligned}
u_1^* &= \min\left\{\max\left\{0, \frac{(\lambda_2 - \lambda_1)f(I)SI}{w_1}\right\}, u_{1\max}\right\}\\
u_2^* &= \min\left\{\max\left\{0, \frac{(\lambda_1 - \lambda_3)S}{w_2}\right\}, u_{2\max}\right\}\\
u_3^* &= \min\left\{\max\left\{0, \frac{(\lambda_2 - \lambda_3)I}{w_3}\right\}, u_{3\max}\right\}
\end{aligned}$$

7. Optimal Control Strategies and Numerical Simulations

In this section, the optimal control strategies to limit disease spread in the community is numerically illustrated by applying the Runge-Kutta forward-backward sweep method to simulate the effects of seven different optimal control strategies on the total infected population and entire susceptible population. The seven different control strategies that are applied for the numerical simulations of the optimal control problem are described as follows:

(i) **Control strategy A:** the optimal use of educational campaign only ($u_1 \neq 0, u_2 = 0, u_3 = 0$).

(ii) **Control strategy B:** the optimal use of vaccination only ($u_2 \neq 0, u_1 = 0, u_3 = 0$).

(iii) **Control strategy C:** the optimal use of treatment only ($u_3 \neq 0, u_1 = 0, u_2 = 0$).

(iv) **Control strategy D:** the optimal use of educational campaign and vaccination only ($u_1 \neq 0, u_2 \neq 0, u_3 = 0$).

(v) **Control strategy E:** the optimal use of educational campaign and treatment only ($u_1 \neq 0, u_3 \neq 0, u_2 = 0$).

(vi) **Control strategy F:** the optimal use of vaccination and treatment only ($u_2 \neq 0, u_3 \neq 0, u_1 = 0$).

(vii) **Control strategy G:** the optimal use of all the control strategies ($u_1 \neq 0, u_2 \neq 0, u_3 \neq 0$).

For numerical simulation, we use a set of parameter values as in the literature [14]: $r = 5, d = 0.02, \delta = 0.01, \alpha = 0.1, \gamma = 0.05, \beta_1 = 0.002$, and $\beta_2 = 0.0018$. Moreover, due to the lack of the available literature and data, we assume the weight values selected for the simulation are $w_1 = 0.001, w_2 = 0.34, w_3 = 0.45$, and $w_4 = 100$. While the initial value of $(S(0), I(0), R(0))$ are assumed to be $(100, 10, 5)$. It should be noted that the weights and the initial values are selected only for the theoretical sense to describe the control strategy proposed in this model. For the maximum control for educational campaign u_1, we set $u_{1\max} = 0.5$ under the assumption that it is difficult to maintain community discipline in implementing preventative strategies such as restrictions on community interaction, and local lockdown. While for the control with vaccination, $u_{2\max} = 0.7$ was taken based on the assumption that the vaccine was not yet fully effective and the lack of awareness of the individual to be vaccinated. However, we assume the maximum control for treatment, $u_{3\max} = 1$ under the assumption that all infected the treatment is fully effective. Next we study the impact of implementing various strategies on the size of infected and susceptible populations for $T = 50$ days.

Strategy A: we demonstrate here the effect of educational campaign u_1 only on the total infected population and entire susceptible population, see Figure 1. In Figure 1a, we notice drastic decrease in the size of the total infected population and similarly in Figure 1b, we observe

decline in the susceptible population size in the presence of control Strategy A when compared with the case without control. We expect to see this type of dynamic since the educational campaign is targeted to educate the population on how to utilize preventive strategies such as keeping social distance, personal hygiene, sanitation and lockdown. These preventive strategies have been shown to reduce the spread of disease. The control profile of Strategy A is depicted in Figure 1c. The result tells us that Strategy A can be implemented by maintaining the educational campaign at the maximum bound till the control intervention period reaches its end.

Strategy B: the effect of of vaccination administration u_2 only, see Figure 2. In Figure 2a and 2b, respectively, we observe the size of both infected population and susceptible population decrease rapidly when compared with the case without control. This result can be used to inform the public about the importance of taking vaccination. The control profile of Strategy C is presented in Figure 2c. The vaccination remains at the maximum value for about 41 days and later drops to zero.

Strategy C: we present here the effect of applying treatment u_3 as depicted in Figure 3. In Figure 3a, we observe rapid decline in the infected population size in the presence of control u_3. Furthermore, Figure 3b depicts that the presence of Strategy C leads to decrease in the susceptible population size but not as much as in the infected population. Figure 3c depicts that the control profile of the treatment is maintained at the maximum bound in the first 8 days and later drops to zero. Thus, we conclude that the treatment can be applied to the host to infected individuals to reduce the disease spread.

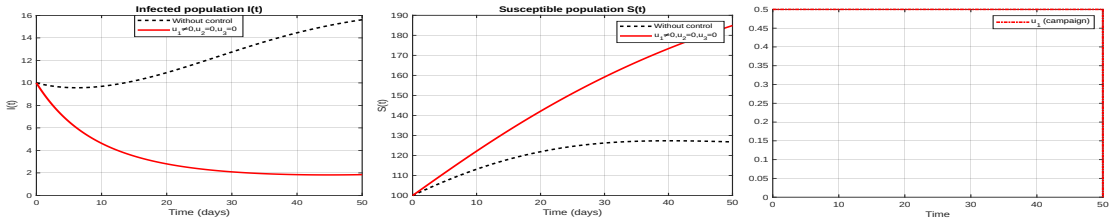
Strategy D: Figure 4 depicts the combined effect of educational campaign u_1, and vaccination u_2 only. The results observed here are similar to the ones depicted in Strategy B. The control profile of Strategy D depicted in Figure 4c. It suggests that the educational campaign u_1 should be maintained at the upper bound for the first 34 days, while the use of vaccination control u_2 should be kept at the maximum value throughout the 50 days. This result inform us that keeping the vaccination at value of 0.5 and promoting the awareness on prevention strategies for most of the time , we could have a better control on the disease spread.

Strategy E: The combined effect of educational campaign u_1, and treatment u_3 only is depicted

in Figure 5. The results observed here are similar to the ones depicted in Strategy A. The respective control profile of the two combined strategies is depicted in Figure 5c. The control profiles of educational campaign u_1 remains at the maximum value for the first 12 days and later drops to zero while the treatment control u_3 is sustained for just 8 days before declines rapidly to zero.

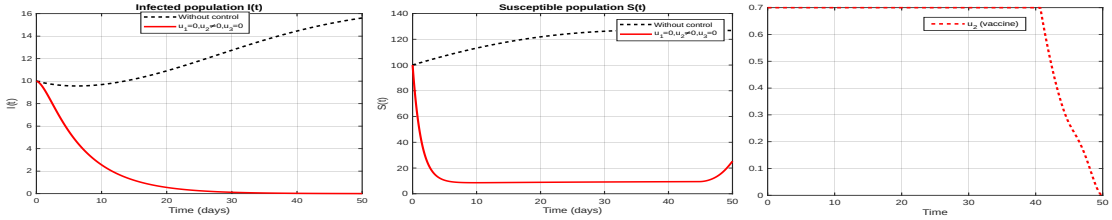
Strategy F: we present here the combined effect of vaccination u_2, and treatment u_3 only on the size of infected and susceptible population, see Figure 7. The results observed here are similar to the ones depicted in Strategy B. The control profiles of Strategy F is depicted in Figure 6c. The vaccination u_2 remains at the maximum value for just 4 days and later drops to zero while the treatment control u_3 is sustained for just 8 days before drops to zero.

Strategy G: we present here the combined effect of utilizing the three controls: educational campaign, vaccination, and treatment on the size of infected and susceptible populations, see Figure 7. We observe sharp drop in both of the infected and susceptible populations as depicted in Figures 7a, and 7b, respectively which is similar to the impact of strategies B and F on the size of the infected and susceptible populations. profile of Strategy G is depicted in Figure 7c. This strategy can be implemented by maintaining the optimal control u_1, u_2 and u_3 at their upper bounds for about 8 days, 4 days, and 6 days respectively, and later all controls decrease rapidly to their lower bounds. When these controls are implemented on a broad scale, it is also critical to adopt an approach that provides optimal cost, i.e., less cost. As a result, we will look at the cost-effectiveness of these controls in the next section [19].



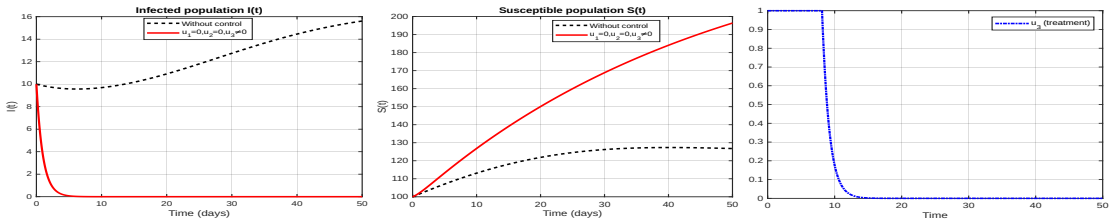
(A) (B) (C)

FIGURE 1. Strategy A optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



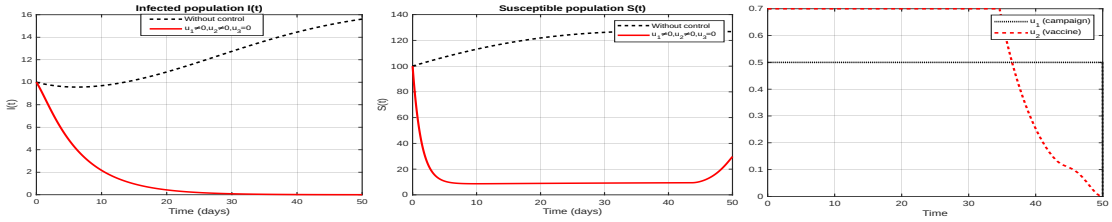
(A) (B) (C)

FIGURE 2. Strategy B optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



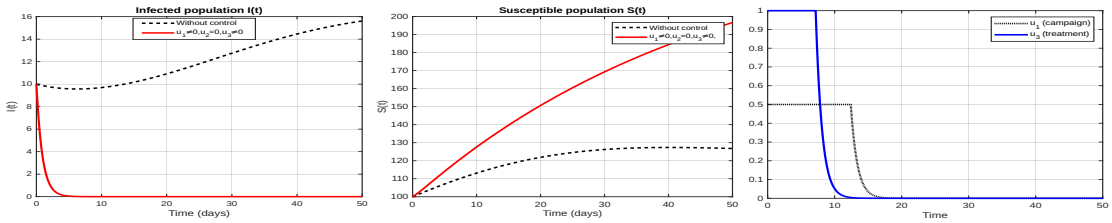
(A) (B) (C)

FIGURE 3. Strategy C optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



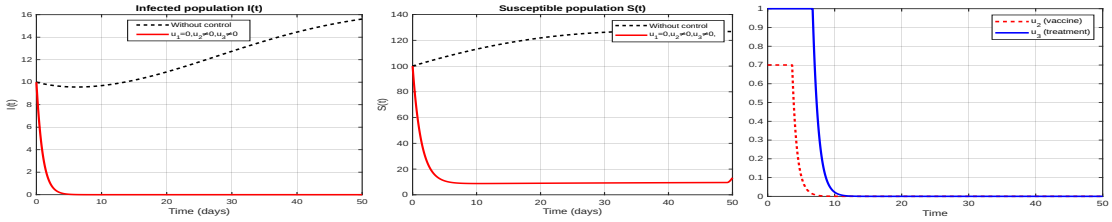
(A) (B) (C)

FIGURE 4. Strategy D optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



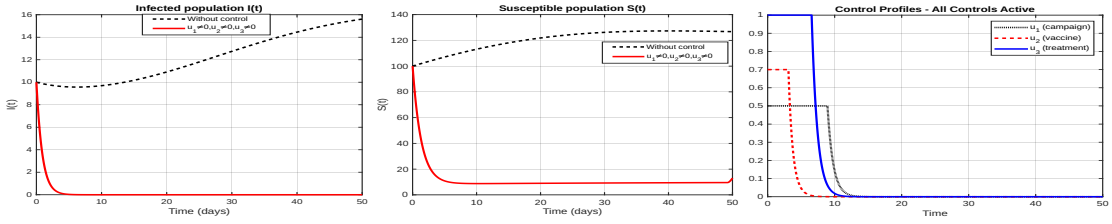
(A) (B) (C)

FIGURE 5. Strategy E optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



(A) (B) (C)

FIGURE 6. Strategy F optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.



(A) (B) (C)

FIGURE 7. Strategy G optimal control on (a) size of infected population $I(t)$; (b) size of susceptible population $S(t)$; and (c) control profile.

8. COST-EFFECTIVENESS ANALYSIS

Cost-effectiveness analysis is a tool used to determine which control strategy with minimal cost. In this section, we use the Average Cost-Effectiveness Ratio (ACER) and the Incremental Cost-Effectiveness Ratio (ICER) to carry out the cost-effectiveness analysis. The Average Cost-Effective Ratio (ACER) is calculated as follows [19]:

$$ACER = \frac{\textit{The total cost } T_c}{\textit{Total number of infections averted } T_a}. \tag{8.1}$$

The total number of individuals infected averted during the intervention period T is obtained by using

$$T_a = \int_0^T (I^*(t) - I(t))\, dt, \tag{8.2}$$

where I^* is the solution to 2.1, without controls, and I the solution to 5.1, with controls. The cost implemented during the period T is calculated as follows

$$T_c = \int_0^T [\frac{1}{2}w_1u_1^2 + \frac{1}{2}w_2u_2^2 + \frac{1}{2}w_3u_3^2]\, dt. \tag{8.3}$$

Based on this cost analysis, the most cost-effective strategy is the one with the smallest ACER value [19]. To analyze the Average Cost-Effectiveness (ACER), the total cost invested and total infected averted in each strategy are calculated. we find that Strategy A has the smallest ACER value and Strategy B has the largest ACER value, as seen in Figure 4. The results are also given in Table 2. Thus, according to the ACER value, the most effective intervention strategy is Strategy A.

The ICER, on the other hand, is calculated by using the following formula[18, 19]

$$ICER = \frac{\textit{Difference in total costs between control strategies}}{\textit{Difference in total infection averted by control strategies}}. \tag{8.4}$$

The difference between the total number of infected individuals without controls and the total number of infected individuals with controls is used to compute the total infection averted . Furthermore,we employed the cost functions $\frac{1}{2}w_1u_1^2 + \frac{1}{2}w_2u_2^2 + \frac{1}{2}w_3u_3^2$ across time to calculate the total cost of the implemented strategies [19]. We also used the parameter values from the preceding section to calculate the total cost and total infections averted, as shown in Table 2,

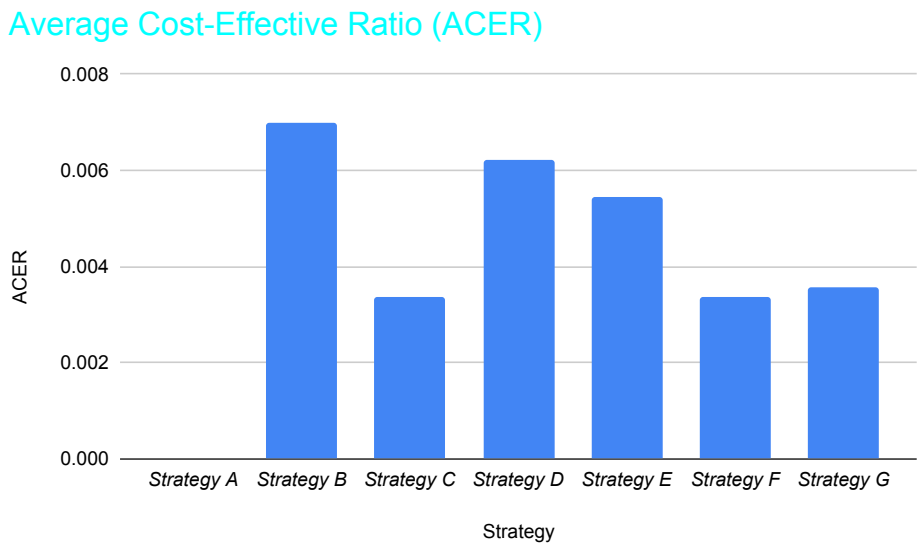


FIGURE 8. Average cost-effectiveness ratio (ACER) results for Strategy A-G

with total infection averted are ranked in ascending order. First, we compute the ICER ratio for the competing strategies A and B as follows:

$$ICER(A) = \frac{0.00625 - 0}{438.798 - 0} = 0.0000142435$$
$$ICER(B) = \frac{3.69243 - 0.00625}{528.672 - 438.798} = 0.0410149$$

It is clearly that Strategy A has the smallest ICER value, Table 2. Therefore, Strategy A which is educational campaign is more effective than Strategy B. As a result, using Vaccine intervention control u_2 alone is more expensive and ineffective than implementing educational campaign control u_1 . As a result, Strategy B is removed from the list of possible control strategies. Next, The ICER for Strategies A and D calculated as follows:

$$ICER(A) = \frac{0.00625 - 0}{438.798 - 0} = 0.0000142435$$
$$ICER(D) = \frac{3.34472 - 0.00625}{537.565 - 438.798} = 0.0338014722$$

t is clearly shown from Table 3 that Strategy D has an ICER value greater than Strategy A. Due to the cost-effectiveness of Strategy D, the combination of educational campaign and vaccination strategy, is removed from the list. To compare between ICER ratios for the remaining

combinations of strategies, we follow the same proceeding approach. Tables 4,5,6,7. summaries the results of ICRE ratios of Strategies A-C, A-F, and A-E, respectively. We observe that Strategy A is the most cost-effectiveness compared to the other possible options.

TABLE 2. ACER of Strategies A-G with the number of averted infected in increasing order

Strategy	Cost	Cases Averted	ACER	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435	0.0000142435
B: $u1 = 0, u3 = 0$	3.69243	528.672	0.00698435	0.0410149
D: $u3 = 0$ only	3.34472	537.565	0.00622198	-
C: $u1 = 0, u2 = 0$	1.99364	594.147	0.00335546	-
F: $u1 = 0$ only	2.0133	594.842	0.00338459	-
E: $u2 = 0$ only	3.24557	594.989	0.00545484	-
G: All controls	2.12962	595.284	0.00357748	-

TABLE 3. Comparison between Strategies A and D

Strategy	Cost	Cases Averted	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435
D: $u3 = 0$ only	3.34472	537.565	0.0338014722

TABLE 4. Comparison between Strategies A and C

Strategy	Cost	Cases Averted	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435
C: $u1 = 0, u2 = 0$	1.99364	594.147	0.01279306593

TABLE 5. Comparison between Strategies A and F

Strategy	Cost	Cases Averted	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435
F: $u1 = 0$ only	2.0133	594.842	0.01286207736

TABLE 6. Comparison between Strategies A and E

Strategy	Cost	Cases Averted	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435
E: $u2 = 0$ only	3.24557	594.989	0.02073947922

TABLE 7. Comparison between Strategies A and E

Strategy	Cost	Cases Averted	ICER
A: $u2 = 0, u3 = 0$	0.00625	438.798	0.0000142435
G: All controls	2.12962	595.284	0.01356907327

9. MAIN RESULTS

We have successfully prove that the system 2.1 is globally asymptotically stable about the endemic equilibrium E^*, if the basic reproduction number $R_0 > 1$. Furthermore, we perform the sensitive analysis on R_0. Both parameters β_1 and r have positive sensitivity indices while the parameters d, α and γ have negative sensitivity indices. We investigate the optimal control model by integrating three-time dependent control variables into the proposed SIRS model 2.1: educational campaign, vaccination and treatment. Seven strategies A-G of different combinations of the three controls are proposed to analyze the impacts of controls on the sizes of infected and the susceptible populations. When compared to the scenario with no control strategy, the results reveal that each of the control strategies is effective in reducing the overall number of infected and susceptible populations. To determine the most cost-effective optimal control strategy, the ACER and ICER are calculated. The study shows that strategy A is the most cost-effective optimal control method which can be used to reduce the spread of disease.

FUNDING

The author declares that this research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

CONFLICT OF INTERESTS

The author declares that there is no conflict of interests.

REFERENCES

[1] C.H. Li, C.C. Tsai, S.Y. Yang, Analysis of epidemic spreading of an SIRS model in complex heterogeneous networks, Commun. Nonlinear Sci. Numer. Simul. 19 (2014), 1042–1054. https://doi.org/10.1016/j.cnsns.2013.08.033.

[2] J. Tolles, T. Luong, Modeling epidemics with compartmental models, JAMA 323 (2020), 2515–2516. https://doi.org/10.1001/jama.2020.8420.

[3] K.M.A. Kabir, K. Kuga, J. Tanimoto, Analysis of SIR epidemic model with information spreading of awareness, Chaos Solitons Fractals 119 (2019), 118–125. https://doi.org/10.1016/j.chaos.2018.12.017.

[4] T. Harko, F.S.N. Lobo, M.K. Mak, Exact analytical solutions of the susceptible-infected-recovered (SIR) epidemic model and of the SIR model with equal death and birth rates, Appl. Math. Comput. 236 (2014), 184–194. https://doi.org/10.1016/j.amc.2014.03.030.

[5] Y. Liu, J.A. Cui, The impact of media coverage on the dynamics of infectious disease, Int. J. Biomathemat. 1 (2008), 65–74. https://doi.org/10.1142/S1793524508000023.

[6] Y. Cheng, J. Wang, X. Yang, On the global stability of a generalized cholera epidemiological model, J. Biol. Dyn. 6 (2012), 1088–1104. https://doi.org/10.1080/17513758.2012.728635.

[7] H.I. Freedman, S. Ruan, M. Tang, Uniform persistence and flows near a closed positively invariant set, J. Dyn. Differ. Equ. 6 (1994), 583–600. https://doi.org/10.1007/BF02218848.

[8] M.A. Khan, Y. Khan, S. Khan, S. Islam, Global stability and vaccination of an SEIVR epidemic model with saturated incidence rate, Int. J. Biomathemat. 9 (2016), 1650068. https://doi.org/10.1142/S1793524516500686.

[9] P. Van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6.

[10] Y.M. Rangkuti, A. Landong, Sensitivity analysis of SEIR epidemic model of COVID-19 spread in Indonesia, J. Phys. Conf. Ser. 2193 (2022), 012092. https://doi.org/10.1088/1742-6596/2193/1/012092.

[11] E. Vynnycky, A. Trindall, P. Mangtani, Estimates of the reproduction numbers of Spanish influenza using morbidity data, Int. J. Epidemiol. 36 (2007), 881–889. https://doi.org/10.1093/ije/dym071.

[12] M.Y. Li, J.S. Muldowney, Geometric approach to global-stability problems, SIAM J. Math. Anal. 27 (1996), 1070–1083. https://doi.org/10.1137/S0036141094266449.

[13] J. Cui, Y. Sun, H. Zhu, The impact of media on the control of infectious diseases, J. Dyn. Differ. Equ. 20 (2008), 31–53. https://doi.org/10.1007/s10884-007-9075-0.

[14] W. Liu, A SIRS epidemic model incorporating media coverage with random perturbation, Abstr. Appl. Anal. 2013 (2013), 792308. https://doi.org/10.1155/2013/792308.

[15] Y. Xiao, T. Zhao, S. Tang, Dynamics of an infectious disease with media/psychology induced non-smooth incidence, Math. Biosci. Eng. 10 (2013), 445–461. https://doi.org/10.3934/mbe.2013.10.445.

[16] P. Muthu, B. Modak, Within-host models of dengue virus transmission with immune response, Comput. Math. Biophys. 11 (2023), 20220150. https://doi.org/10.1515/cmb-2022-0150.

[17] M. Barro, A. Guiro, D. Ouedraogo, Optimal control of a SIR epidemic model with general incidence function and time delays, Cubo (Temuco) 20 (2018), 53–66. http://dx.doi.org/10.4067/S0719-06462018000200053.

[18] M.M. Ojo, T.O. Benson, A.R. Shittu, E.F.D. Goufo, Optimal control and cost-effectiveness analysis for the dynamic modeling of Lassa fever, J. Math. Comput. Sci. 12 (2022), Article-ID. https://doi.org/10.28919/jmcs/7279.

[19] Y.A. Adi, N. Irsalinda, M. Z., Optimal control and cost-effectiveness analysis in an epidemic model with viral mutation and vaccine intervention, Cauchy J. Math. Murni Apl. 7 (2022), 173–185. https://doi.org/10.1016/j.physa.2022.127804.

[20] Reginald Davey, What is Epidemiologic Modeling? https://www.news-medical.net/health/What-is-Epidemiologic-Modeling.aspx.

[21] P.J. White, Mathematical models in infectious disease epidemiology, Infect. Dis. (2017), 49–53.e1. https://doi.org/10.1016/b978-0-7020-6285-8.00005-8.

[22] J.K. Hale, Ordinary differential equations, John Wiley, New York, 1969.

[23] P. Hartman, Ordinary differential equations, John Wiley, New York, 1965.