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STABILITY AND OPTIMAL CONTROL ANALYSIS OF AN SCIR EPIDEMIC MODEL

OLUMUYIWA JAMES PETER[1,*], RATCHADA VIRIYAPONG[2], FESTUS ABIODUN OGUNTOLU[3], PENSIRI YOSYINGYONG[2], HELEN OLARONKE EDOGBANYA[4], MICHAEL OYELAMI AJISOPE[5]

[1]Department of Mathematics, University of Ilorin, Ilorin, Kwara State, Nigeria

[2]Department of Mathematics, Faculty of Science, Naresuan University, Phitsanulok, Thailand

[3]Department of Mathematics, Federal University of Technology Minna, Minna, Niger State, Nigeria

[4]Department of Mathematical Sciences, Federal University Lokoja, Lokoja, Kogi State, Nigeria

[5]Department of Mathematics, Federal University Oye-Ekiti, Ekiti State, Nigeria

Abstract: In this paper, we proposed a deterministic model of *SCIR* governed by a system of nonlinear differential equations. Two equilibria (disease-free and endemic) are obtained and the basic reproduction number R_0 is calculated. If R_0 is less than one, then the disease-free equilibrium state is globally stable i.e. the disease will be eradicated eventually. However, when R_0 is greater than unity, the disease persists and the endemic equilibrium point is globally stable. Furthermore, the optimal control problem is applied into the model. The focus of this study is to determine what control method can be implemented to significantly slow the incidence of the epidemic disease, therefore we take into account various possible combinations of such three controls which are prevention via proper hygiene, screening of the infected carriers which enable them to know their health conditions and to go for early treatment and treatment of the infected individuals. The possible strategies of using combinations of the three controls on the spread of the disease, one at a time or two at a time is also discussed. Our numerical analysis of the optimal approach suggests that the best method is to incorporate all three controls in order to control the disease epidemic.

*Corresponding author

E-mail address: peterjames4real@gmail.com

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1. INTRODUCTION

Infectious diseases have been the greatest threat to the existence and well-being of human since the time immemorial. While some individuals who are less fortunate to survive the menace of infectious diseases are left with one deformity or the other, tens of thousands of lives across the globe have been lost to the emergence and re-emergence of infectious diseases like cholera [1], typhoid fever [2], HIV/AIDS [3], Ebola [4], Lassa fever [5], measles [6], Tuberculosis [7] and a host of others [8-10]. The dynamics of infectious diseases becomes more complicated and devastating with the existence of asymptomatic nature of some of the diseases (such as typhoid fever, hepatitis B, tuberculosis, Epstein-Barr virus, measles, HIV/AIDS and Clostridium difficile-associated disease) [8]. The complexity of the infectious diseases with asymptomatic feature emanates from the fact that the infected individuals can spread the illness without exhibiting any symptoms [10], some may call carrier. This set of people spread diseases more quickly and with ease.

The havoc of the infectious diseases has made their prevention and management the top priority of the government the world over. Government and policy makers are aided by the results from the researches in formulating and implementing appropriate policies to prevent or curb the outbreak of infectious diseases. Mathematical models have been playing significant roles in quantifying probable infectious disease interventions and mitigation approaches [11-16]. There exists a good number of models for contagious diseases; as regards compartmental models, beginning from the simplest *SIR* model invested by Kermack and Mckendrick in 1927 [17-18], to more complicated models (such as *SEIR*, P_1P_2SEIR, $S_HI_HR_HS_vI_v$, *SVLIT*) [6,19-21].

Mathematical modelling of infectious diseases applying optimal control theory has been widely studied in the literature. Particularly, the popular Pontryagin's maximum principle pioneered by Pontryagin and co-researchers [22] and later improved by Fleming and Rishel [23] has been applied in many studies to examine optimal control in a good number of models of epidemic diseases including pandemic influenza, HIV diseases and vector borne infections [24-33]. Okosun et al. [27] examine the impact of optimal control to analyze the role of screening and treatment of asymptomatic individuals on HIV/AIDS dynamics. Roy et al. [29] investigate the impact of education programs in curtailing the HIV/AIDS transmission applying an optimal

control theory.

Tchuenche et al. [30] design a model to explore the role of media on the dynamics of influenza and the cost implication of implementing education, vaccination and the media in fighting the spread of influenza using the theory of optimal control. Also, Mistra et al. [31] design an *SIS* epidemic model and investigate the role of media on the dynamics of diseases that transmit from human-to-human interactions adopting an optimal control theory. A detailed literature of the theory of optimal control in epidemic models and numerical approximation methods exists in [34-35]. Several studies in the literature confirm that infectious disease models which are developed with the theory of optimal control are suitable and extremely useful for predicting intervention strategies to control disease transmission [36-47]. Our goal is to propose a general mathematical model for infectious diseases that includes carrier individuals which is suitable for the ones mentioned in the literature with application of optimal control and to investigate the effects of prevention via proper hygiene, screening of the infected carriers, and treatment of the infected individuals on the disease transmission dynamics which has not been considered in the previous study.

The study considers control interventions for dynamics of an epidemic disease and to establish interventions that are vital in bringing the spread and transmission of an infectious disease under control. The paper is organized as follows, the model formulation with description is presented in section 2. Model analysis including the boundary of the solution, basic reproduction number, equilibrium points and their global stability and sensitivity analysis is demonstrated in section 3. Section 4 shows how the model is extended to optimal control model where its numerical simulations are presented in section 5. Finally, a brief discussion and conclusion is in section 6.

2. MATERIALS AND METHOD

2.1 Formulation of the model

We formulate an *SCIR* deterministic model to study the transmission dynamics of infectious disease. The population under consideration consist of four compartments, susceptible class *S(t),* carrier infected class *C(t),* infected class *I(t)* and recovered class *R(t),* respectively. Susceptible population increases as a result of daily recruitment by birth or immigration at the rate π and it is reduced as a result of natural mortality rate μ. Susceptible individual can be infected through direct contact with the asymptomatic carrier without symptoms with a probability ρ and

with symptomatic infected individuals with a probability $1-\rho$. It is assumed that the disease transmission rate α for the carrier individual is greater than the disease transmission rate of infected individuals β this is because those in carrier class are equally infectious but they are not aware they are infected with the disease. The rate at which those in the carrier show symptoms is denoted by θ. Infected individuals recovered through the rate γ and lose immunity after some times and upon recovery become susceptible again. The flow chart and the description of the parameters are illustrated in Figure 1.

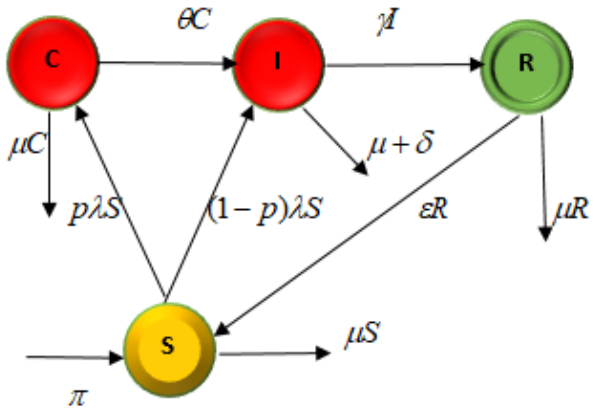


FIGURE 1. The flow chart of *SCIR* model.

The following set of nonlinear ordinary differential equations can be obtained from the definition and the compartmental diagram in Figure 1 above:

$$
\begin{aligned}
\frac{dS}{dt} &= \pi - (\lambda + \mu)S + \varepsilon R \\
\frac{dC}{dt} &= p\lambda S - (\mu + \theta)C \\
\frac{dI}{dt} &= (1-p)\lambda S - (\mu + \gamma + \delta)I + \theta C \\
\frac{dR}{dt} &= \gamma I - (\mu + \varepsilon)R
\end{aligned} \tag{1}
$$

where $\lambda = (\alpha C + \beta I)$.

3. ANALYSIS OF THE MODEL

3.1. Boundedness of the solution

First, we let the total population be $M(t) = S(t) + C(t) + I(t) + R(t)$. Then,

$$M' = S' + C' + I' + R' = \pi - \delta I - \mu M \,. \tag{2}$$

Therefore,

$$\frac{dM}{dt} \le \pi - \mu M \,. \tag{3}$$

Integrating both side of (3), we have

$$\int_0^t \frac{dM}{\pi - \mu M} \le \int_0^t dt \quad ,$$

$$-\frac{1}{\mu}\ln(\pi - \mu M) \le t \quad . \tag{4}$$

From (4), we have

$$M \le \frac{\pi}{\mu} - \left[\frac{\pi - \mu M_0}{\mu}\right] e^{-\mu t} ,$$

by taking $t \to \infty$, then we obtain that $M \le \frac{\pi}{\mu}$.

This implies that the model in (1) can be studied in the feasible region

$$\Omega = \left\{ (S, C, I, R) \in R_+^4 : M \le \frac{\pi}{\mu} \right\} \tag{5}$$

3.2. Positivity of the solution

Theorem 1: Given $S_0 > 0, C_0 > 0, I_0 > 0, R_0 > 0$ then the solution $\{(S, C, I, R) \in R_+^4\}$ are non-negative invariant for $t \ge 0$

Proof.

Recall from (1),

$$\frac{dS}{dt} = \pi - (\lambda + \mu)S + \varepsilon R$$

$$\frac{dS(t)}{dt} \ge -(\lambda + \mu)S \,.$$

By separating the variable and integrating inequality above, we have

$$\int_0^t \frac{1}{S}\, dS \geq \int_0^t -(\lambda+\mu)dt\,.$$

Thus,

$$S(t) \geq S_0 e^{-(\mu+\lambda)t} \geq 0\,. \tag{6}$$

By following the same process, we obtain

$$C(t) \geq C_0 e^{-(\mu+\theta)t} \geq 0$$

$$I(t) \geq I_0 e^{-(\mu+\gamma+\delta)t} \geq 0 \tag{7}$$

$$R(t) \geq R_0 e^{-(\mu+\varepsilon)t} \geq 0$$

Hence, the solution of (1) is non-negative. □

3.3. Disease-free equilibrium (DFE)

This occurs in the absence of infection. Thus, in the absence of infection, we set C and I to zero and the resulting solution gives the disease-free equilibrium states given as

$$\Phi_{DFE}(S,C,I,R) = \left(\frac{\pi}{\mu},0,0,0\right)\,. \tag{8}$$

3.4. Endemic equilibrium

This occurs when the infection persists in the population represented by $\Phi_{EE}(S^*,C^*,I^*,R^*)$. To obtain it, we set the LHS of (1) to zero. Thus,

$$S^* = \frac{k_1 k_3}{\alpha p k_3 + \beta k_1 k_2 + \beta p\theta}$$

$$C^* = \frac{k_1 k_4 p(k_1 k_3 \mu - \pi\alpha k_3 p - \pi\beta k_1 k_2 - \pi\beta p\theta)}{\alpha\varepsilon\gamma k_1 k_2 k_3 p + \alpha\varepsilon\gamma k_3 p^2\theta + \beta\varepsilon\gamma k_1^2 k_2^2 + 2\beta\varepsilon\gamma k_1 k_2 p\theta + \beta\varepsilon\gamma p^2\theta^2 - \alpha k_1 k_3^2 k_4 p - \beta k_1^2 k_2 k_3 k_4 - \beta k_1 k_3 k_4}$$

$$I^* = \frac{k_4(k_1 k_3 \mu - \pi\alpha k_3 p - \pi\beta k_1 k_2 - \pi\beta p\theta)(k_1 k_2 + p\theta)}{(\alpha k_3 p + \beta k_1 k_2 + \beta p\theta)(\varepsilon\gamma k_1 k_2 + \varepsilon\gamma p\theta - k_1 k_3 k_4)}$$

$$R^* = \frac{\gamma(k_1 k_3 \mu - \pi\alpha k_3 p - \pi\beta k_1 k_2 - \pi\beta p\theta)(k_1 k_2 + p\theta)}{(\alpha k_3 p + \beta k_1 k_2 + \beta p\theta)(\varepsilon\gamma k_1 k_2 + \varepsilon\gamma p\theta - k_1 k_3 k_4)},$$

where $k_1 = \mu+\theta$, $k_2 = 1-p$, $k_3 = \mu+\gamma+\delta$ and $k_4 = \mu+\varepsilon$.

3.5. The basic reproduction number

The basic reproductive ratio is a threshold quantity that shows the total number of possible diseases due to a single infected individual, generated throughout its contagious period into a fully susceptible population. *F* and *V* are the matrices for the new infections generated and the terms of transition, respectively. Following the same approach as [27], we determine the basic reproduction number as follows.

The new infection compartments are *C(t)* and *I(t)*,

$$\frac{dC}{dt} = pS(\alpha C + \beta I) - (\mu + \theta)C$$
$$\frac{dI}{dt} = (1-p)S(\alpha C + \beta I) - (\mu + \gamma + \delta)I + \theta C$$

$$f = \begin{pmatrix} pS(\alpha C + \beta I) \\ (1-p)S(\alpha C + \beta I) \end{pmatrix}, \quad v = \begin{pmatrix} (\mu + \theta)C \\ (\mu + \gamma + \delta)I - \theta C \end{pmatrix}.$$

F and *V* are the Jacobian matrix which shall be computed at the DFE such that,

$$F = \begin{pmatrix} pS\alpha & pS\beta \\ k_2\alpha S & k_2\beta S \end{pmatrix}, \quad V = \begin{pmatrix} k_1 & 0 \\ -\theta & k_3 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 \\ \frac{\theta}{k_1 k_3} & \frac{1}{k_3} \end{pmatrix}$$

where $k_1 = \mu + \theta$, $k_2 = 1 - p$, $k_3 = \mu + \gamma + \delta$.

Thus, $FV^{-1} = \begin{bmatrix} \frac{p\alpha S}{k_1} + \frac{p\beta S\theta}{k_1 k_3} & \frac{p\beta S}{k_3} \\ \frac{k_2\alpha S}{k_1} + \frac{k_2\beta S\theta}{k_1 k_3} & \frac{k_2\beta S}{\beta S} \end{bmatrix}$.

The spectral radius of the matrix ρFV^{-1} which is the basic reproduction number R_0 is obtained as

$$R_0 = \frac{\pi\left(\alpha k_3 p + \beta k_1 k_2 + \beta p\theta\right)}{\mu k_1 k_3} \tag{9}$$

3.6. Global stability of the disease-free equilibrium

Theorem 2: The disease-free equilibrium (DFE) Φ_{DFE} of model (1) is globally asymptotically stable, when $R_0 < 1.$

Proof: To investigate the global asymptotic stability of the DFE, we apply the process of Lyapunov functions. First, we define a Lyapunov function L as follows:

$$L(t) = \left(\frac{\alpha}{\mu+\theta} + \frac{\beta\theta}{(\mu+\delta+\gamma)(\mu+\theta)}\right)C + \left(\frac{\beta}{\mu+\delta+\gamma}\right)I. \tag{10}$$

Then, differentiating L along with the solutions of (1) gives

$$\begin{aligned}
\frac{dL(t)}{dt} &= \left(\frac{\alpha}{\mu+\theta} + \frac{\beta\theta}{(\mu+\delta+\gamma)(\mu+\theta)}\right)\left(pS(\alpha C+\beta I) - (\mu+\theta)C\right) \\
&+ \left(\frac{\beta}{\mu+\delta+\gamma}\right)\left((1-p)S(\alpha C+\beta I) - (\mu+\gamma+\delta)I + \theta C\right) \\
&= \left(\frac{p\alpha(\mu+\delta+\gamma) + p\beta\theta + (1-p)\beta(\mu+\theta)}{(\mu+\delta+\gamma)(\mu+\theta)}\right)S(\alpha C+\beta I) \\
&- \alpha C - \frac{\beta\theta C}{\mu+\delta+\gamma} - \beta I + \frac{\beta\theta C}{\mu+\delta+\gamma} \\
&\le \left(\frac{\pi\left(p\alpha(\mu+\delta+\gamma) + (1-p)\beta(\mu+\theta) + p\beta\theta\right)}{\mu(\mu+\delta+\gamma)(\mu+\theta)} - 1\right)(\alpha C+\beta I) \\
&= (R_0 - 1)(\alpha C+\beta I).
\end{aligned}$$

We obtain that $\frac{dL(t)}{dt} = 0$ when $C = I = 0$ and $\frac{dL(t)}{dt} < 0$, when $R_0 < 1$. By Lyapunov-LaSalle theorem [48], we can conclude that the disease-free equilibrium (DEF) Φ_{DEF} is globally asymptotically stable, when $R_0 < 1.$ This completes the proof.

3.6. Global stability of the endemic equilibrium

The global stability of the endemic equilibrium is investigated by using the geometric approach of Li and Muldowney [49]. First, we need to start by proving the following lemma.

Lemma 3. The system (1) is uniformly persistent if and only if $R_0 > 1$

Proof. We obtain earlier that whenever $R_0 > 1$, the disease-free equilibrium is unstable. With the result by [50]. (Theorem 4.3), this result obtained is equivalence to the uniform persistence of

system (1). Therefore, the system (1) is uniformly persistent if and only if $R_0 > 1$ i.e. there exists a constant $z > 0$ such that,

$$\liminf_{t\to\infty} S(t) \geq z,\ \liminf_{t\to\infty} C(t) \geq z,\ \liminf_{t\to\infty} I(t) \geq z,\ \liminf_{t\to\infty} R(t) \geq z$$

provided that $(S(0), C(0), I(0), R(0)) \in \Omega$.

Further, the uniform persistence of the state variable together with boundedness of Ω is equivalent to the existence of a compact absorbing set in Ω.

Theorem 3: The endemic equilibrium point $\Phi_{EE}(S^*, C^*, I^*, R^*)$ is globally asymptotically stable in Ω when $R_0 > 1$ and $\bar{b} > 0$ ($\bar{b}$ is defined in the proof).

Proof. We first write the Jacobian matrix of subsystem of (1), we have

$$J(S,C,I) = \begin{pmatrix} -\alpha C - \beta I - \mu & -\alpha S & -\beta S \\ p(\alpha C + \beta I) & p\alpha S - \mu - \theta & p\beta S \\ (1-p)(\alpha C + \beta I) & (1-p)\alpha S + \theta & (1-p)\beta S - \mu - \gamma - \delta \end{pmatrix}. \tag{11}$$

Then, its second additive compound matrix is given by

$$J^{[2]} = \begin{pmatrix} -\alpha C - \beta I + p\alpha S - 2\mu - \theta & p\beta S & \beta S \\ (1-p)\alpha S + \theta & -\alpha C - \beta I + (1-p)\beta S - 2\mu - \gamma - \delta & -\alpha S \\ -(1-p)(\alpha C + \beta I) & p(\alpha C + \beta I) & p\alpha S - 2\mu - \theta + (1-p)\beta S - \gamma - \delta \end{pmatrix} \tag{12}$$

Next, set the function $P = diag\left(1, \frac{C}{I}, \frac{C}{I}\right)$. Then,

$$P_f P^{-1} = diag\left(0, \frac{C'}{C} - \frac{I'}{I}, \frac{C'}{C} - \frac{I'}{I}\right),$$

where the matrix P_f is obtained by replacing each entry p_{ij} of P by its derivative in the direction of solution of subsystem of (1). Further, we consider the matrix $B = P_f P^{-1} + PJ^{[2]}P^{-1}$. The block

form is written as $B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$, where

$$B_{11} = -\alpha C - \beta I + p\alpha S - 2\mu - \theta$$

$$B_{12} = \begin{pmatrix} \frac{p\beta SI}{C} & \frac{\beta SI}{C} \end{pmatrix}, B_{21} = \begin{pmatrix} \left((1-p)\alpha S + \theta\right)\frac{C}{I} \\ \left(-(1-p)(\alpha C + \beta I)\right)\frac{C}{I} \end{pmatrix}$$

$$B_{22} = \begin{pmatrix} -\alpha C - \beta I + (1-p)\beta S - 2\mu - \gamma - \delta + \frac{C'}{C} - \frac{I'}{I} & -\alpha S \\ p(\alpha C + \beta I) & p\alpha S - 2\mu - \theta + (1-p)\beta S - \gamma - \delta + \frac{C'}{C} - \frac{I'}{I} \end{pmatrix}.$$

Let (x_1, x_2, x_3) be a vector in $\Re^3$, we then select a norm in $\Re^3$ that is defined as $|x_1, x_2, x_3| = \max\{|x_1|, |x_2| + |x_3|\}$ and denote v as the *Lozinskii* measure with respect to this norm. With the method of [51], we have an estimation of $v(B)$ as follows:

$$v(B) \le \sup\{h_1, h_2\},$$

where $h_1 = v_1(B_{11}) + |B_{12}|$ and $h_2 = |B_{21}| + v_1(B_{22})$. The v_1 represent the *Lozinskii* measure with respect to l_1 vector norm and $|B_{12}|$ and $|B_{21}|$ are matrix norms with respect to l_1 vector norm. Therefore, for our study we have

$$v(B_{11}) = -\alpha C - \beta I + p\alpha S - 2\mu - \theta \quad , \quad \|B_{21}\| = \left((1-p)\alpha S + \theta\right)\frac{C}{I} + (1-p)(\alpha C + \beta I)\frac{C}{I}$$

$$v(B_{22}) = \max\left\{-\alpha C - \beta I + (1-p)\beta S - 2\mu - \gamma - \delta + \frac{C'}{C} - \frac{I'}{I} + p(\alpha C + \beta I), \quad p\alpha S - 2\mu - \theta + (1-p)\beta S - \gamma - \delta + \alpha S + \frac{C'}{C} - \frac{I'}{I}\right\}.$$

Therefore, we have

$$h_1 = -\alpha C - \beta I + p\alpha S - 2\mu - \theta + \frac{\beta SI}{C}$$

$$h_2 = \left[(1-p)\alpha S + \theta + (1-p)(\alpha C + \beta I)\right]\frac{C}{I} + \max\left\{-\alpha C - \beta I + (1-p)\beta S - 2\mu - \gamma - \delta + \frac{C'}{C} - \frac{I'}{I} + p(\alpha C + \beta I), \quad p\alpha S - 2\mu - \theta + (1-p)\beta S - \gamma - \delta + \alpha S + \frac{C'}{C} - \frac{I'}{I}\right\}.$$

Since from our system we have $C' = p(\alpha C + \beta I)S - (\mu + \theta)C$, then

$$\frac{C'}{C} = p(\alpha C+\beta I)\frac{S}{C}-(\mu+\theta),$$

thus $$-(\mu+\theta) = \frac{C'}{C}-p(\alpha C+\beta I)\frac{S}{C}.$$

Therefore, h_1 becomes

$$h_1=\frac{C'}{C}-p(\alpha C+\beta I)\frac{S}{C}-\alpha C-\beta I+p\alpha S-\mu+\frac{\beta SI}{C} = \frac{C'}{C}-\alpha C-\beta I-\mu+(1-p)\frac{\beta SI}{C}.$$

Similarly, we have $\frac{I'}{I}=(1-p)(\alpha C+\beta I)\frac{S}{I}-(\mu+\gamma+\delta)+\frac{\theta C}{I}$, then

$$-(\mu+\delta+\gamma)=\frac{I'}{I}-(1-p)(\alpha C+\beta I)\frac{S}{I}-\frac{\theta C}{I}.$$

Therefore, h_2 becomes

$$\begin{aligned}h_2=&\left[(1-p)\alpha S+\theta+(1-p)(\alpha C+\beta I)\right]\frac{C}{I}\\&+\max\{-\alpha C-\beta I+(1-p)\beta S-\mu+\frac{I'}{I}-(1-p)(\alpha C+\beta I)\frac{S}{I}-\frac{\theta C}{I}+\frac{C'}{C}-\frac{I'}{I}+p(\alpha C+\beta I),\\&\quad p\alpha S-\mu-\theta+(1-p)\beta S-\gamma-\delta+\alpha S+\frac{I'}{I}-(1-p)(\alpha C+\beta I)\frac{S}{I}-\frac{\theta C}{I}+\frac{C'}{C}-\frac{I'}{I}\}.\end{aligned}$$

Hence, we obtain that

$$v(B)\le \max\{h_1,h_2\}$$

That is

$$v(B)\le \frac{C'}{C}+\max\left\{(1-p)\frac{\beta SI}{C}-\alpha C-\beta I-\mu,(1-p)(\alpha C+\beta I)\frac{C}{I}-\mu+\sup\{-\alpha C-\beta I+p(\alpha C+\beta I),p\alpha S-\theta+\alpha S\}\right\}.$$

And,

$v(B)\le \frac{C'}{C}-\bar{b}$, where

$$\bar{b} = \min\left\{\alpha C+\beta I+\mu-(1-p)\frac{\beta SI}{C},\mu-(1-p)(\alpha C+\beta I)\frac{C}{I}-\inf\{\alpha C+\beta I-p(\alpha C+\beta I),\theta-\alpha S-p\alpha S\}\right\}.$$

Next, we consider any solution $S(t),C(t),I(t)$ emanating from the compact set in $\Gamma\subset\Omega$. Let $\bar{t}$ be large enough such that the system is uniformly persistent for all $t>\bar{t}$. Then along each solution $(S(t),C(t),I(t))$ such that, $(S(0),C(0),I(0))\in\Gamma$, for $t>\bar{t}$, $\frac{1}{t}(\ln C(t)-\ln C(0))<\frac{\bar{b}}{2}$. Consequently,

$$\begin{aligned}\bar{q}_2=\frac{1}{t}\int_0^t v(B)\,ds &\le \frac{1}{t}\int_0^t\left(\frac{C'}{C}-\bar{b}\right)ds\\&=\frac{1}{t}(\ln C(t)-\ln C(0))-\bar{b}\\&<-\frac{\bar{b}}{2}.\end{aligned}\tag{13}$$

Hence, $\bar{q}_2 \le -\frac{\bar{b}}{2} < 0$. By Theorem 3.5 of Li and Muldowney, [51], we can conclude that the endemic equilibrium (S^*, C^*, I^*) is globally asymptotically stable when $R_0 > 1$ and $\bar{b} > 0$.

Next, we consider the fourth equation of system (1) which is

$$\frac{dR}{dt} = \gamma I - (\mu + \varepsilon) R,$$

where its limit system is $\frac{dR}{dt} = \gamma I^* - (\mu + \varepsilon) R$.

With the condition of equilibrium point, we have $\gamma I^* = (\mu + \varepsilon) R^*$, then we can rewrite the fourth equation of system (1) as

$$\frac{dR}{dt} = (\mu + \varepsilon) R^* - (\mu + \varepsilon) R = (\mu + \varepsilon)(R^* - R).$$

Solving above equation by integration, we have

$$\int_0^t \frac{1}{(\mu + \varepsilon)(R^* - R)} dR = \int_0^t ds$$

$$-(\mu + \varepsilon) \ln |R^* - R(t)|_0^t = t$$

$$\ln \frac{|R^* - R(t)|}{|R^* - R(0)|} = \frac{-t}{\mu + \varepsilon}$$

$$R(t) = R^* - (R^* - R(0)) e^{-\frac{1}{\mu + \varepsilon} t}.$$

When taking $t \to \infty$, we have. $R(t) \to R^*$

Therefore, the endemic equilibrium point (S^*, C^*, I^*, R^*) is globally asymptotically stable whenever $R_0 > 1$ and $\bar{b} > 0$. □

3.7. Sensitivity analysis

In this section, the sensitivity analysis of the basic reproduction number is determined. The results of this analysis shows how each parameter within the model affects the disease transmission. The normalized forward sensitivity index method is used to obtain such sensitivity indices [52-53]. The normalized forward sensitivity index of R_0 with respect to a parameter P is denoted by:

(14) $$S_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0}.$$

The sensitivity indices in Table 1 are derived by using parameters values from Table 2. We obtain that the sensitivity indices of π, β, α and p are positive, this means that in order to reduce the basic reproduction number, these parameters values should be reduced. On the contrary, we should try to increase the parameters θ, γ, δ and μ.

Table 1. Numerical values of sensitivity indices of R_0

Parameter	Sensitivity index
π	1.0000
β	0.8873
α	0.1127
p	0.0902
θ	- 0.0827
γ	-0.1972
δ	-0.5127
μ	-1.2074

With our results above, we therefore propose three strategies to help to limit the disease transmission i.e. to encourage to have hygiene care to reduce the transmission rate, to have screening of the infected carriers which enable them to know their health conditions which will decrease the number of infected individuals and to encourage to have treatment to increase the value of γ.

Table 2. Parameters values

Parameter	Value	Source	Parameter	Value	Source
π	100	Assumed	θ	0.2	Assumed
μ	0.018	[15]	α	0.002	Assumed
ε	0.00094	[15]	δ	0.052	Estimated
p	0.3	Assumed	β	0.002	Assumed
γ	0.02	Assumed			

4. OPTIMAL CONTROL SYSTEM

We extend the model in (1) into optimal control by incorporating three control variables namely; prevention via proper hygiene g_1, screening of the infected carriers which enable them to know their health conditions and to go for early treatment g_2 and treatment of the infected individuals g_3. By incorporating the above descriptions into the basic model in (1) we arrived at the following equations

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \mu S - S(\alpha C + \beta I)(1-g_1) + \varepsilon R \\
\frac{dC}{dt} &= pS(\alpha C + \beta I)(1-g_1) - \mu C - (\theta + g_2)C \\
\frac{dI}{dt} &= (1-p)(1-g_1)S(\alpha C + \beta I) + (1-g_2)\theta C - (g_3 + \gamma)I - (\mu + \delta)I \\
\frac{dR}{dt} &= (g_3 + \gamma)I - (\mu + \varepsilon)R
\end{aligned} \quad , \tag{15}$$

where $S \geq 0, C \geq 0, I \geq 0, R \geq 0$.

We intend to minimize the infected carriers C and the infected individuals I and the associated cost of using the control g_1, g_2 and g_3. By following the approach of [22] and [23], the objective function is defined as

$$J(g_1, g_2, g_3) = \int_0^{t_f} \left(X_1 C + X_2 I + C_1 \frac{g_1^2}{2} + C_2 \frac{g_2^2}{2} + C_3 \frac{g_3^2}{2} \right) dt \quad , \tag{16}$$

where X_1 and X_2 are positive weight constant of infected carriers and infected individuals respectively as defined in (16) and C_1, C_2 and C_3 are the associated cost involved with the use of sanitation, treatment via drugs and screening of the infected carriers respectively. The main aim is to obtain the three optimal control sets g_1^*, g_2^*, g_3^* such that,

$$J\left(g_1^*, g_2^*, g_3^*\right) = \min\left\{ J(g_1, g_2, g_3) \middle| g_1, g_2, g_3 \in G \right\}, \tag{17}$$

where

$$G = \left\{ g = (g_1, g_2, g_3),\ g_i(t)\ \textit{is Lebesque Measurable function on}\ \left[0, t_f\right], 0 \leq g_i \leq 1, i = 1, 2, 3 \right\}.$$

We show here the optimal control problem exists. Using the approach of Pontryagin's Maximum Principle [22] and the differential equations in (15), we define the Hamiltonian H as

$$H = J^{'} + \lambda_1 S^{'} + \lambda_2 C^{'} + \lambda_3 I^{'} + \lambda_4 R^{'}$$

$$\begin{aligned} H(S,C,I,R) = & \left(X_1 C + X_2 I + C_1 \frac{g_1^2}{2} + C_2 \frac{g_2^2}{2} + C_3 \frac{g_3^2}{2} \right) \\ & + \lambda_1 \left(\pi - \mu S - S(\alpha C + \beta I)(1-g_1) + \varepsilon R \right) \\ & + \lambda_2 \left(pS(\alpha C + \beta I)(1-g_1) - \mu C - (\theta + g_2) C \right) \\ & + \lambda_3 \left((1-p)(1-g_1) S(\alpha C + \beta I) + (1-g_2)\theta C - (g_3 + \gamma) I - (\mu + \delta) I \right) \\ & + \lambda_4 \left((g_3 + \gamma) I - (\mu + \varepsilon) R \right) \end{aligned} \tag{18}$$

where $\lambda_i(4)$ are the adjoint variable functions.

Theorem 4:

There exist an optimal control set g_1, g_2 and g_3 corresponding to the solution $\left(S(t), C(t), I(t), R(t)\right)$ that minimize $J(g_1, g_2, g_3)$ over G and the adjoint variables $\lambda_1 \ldots\ldots \lambda_4$ such that

$$\lambda_1^{'} = -\lambda_1 \left(-\mu - (\alpha C + \beta I)(1-g_1) \right) - \lambda_2 \left((1-g_1) p(\alpha C + \beta I) \right) - \lambda_3 \left((1-g_1)(1-p)(\alpha C + \beta I) \right)$$

$$\lambda_2^{'} = -\lambda_1 \left(-\alpha S(1-g_1) \right) - \lambda_2 \left((1-g_1) p\alpha S - \mu - (\theta + g_2) \right) - \lambda_3 \left((1-g_1)(1-p)\alpha S + (1-g_2)\theta \right) - X_1$$

$$\lambda_3^{'} = -\lambda_1 \left(-\beta S(1-g_1) \right) - \lambda_2 \left((1-g_1) p\beta S \right) - \lambda_3 \left((1-g_1)(1-p)\beta S - (g_3 + \gamma) - (\mu + \delta) \right) - \lambda_4 (g_3 + \gamma) - X_2$$

$$\lambda_4^{'} = -\lambda_1 \varepsilon + \lambda_4 (\mu + \varepsilon)$$

subject to the transversality conditions,

$\lambda_1(t_f), \ldots\ldots\ldots, \lambda_4(t_f) = 0$.

Furthermore, the optimal control g_1, g_2, g_3 are given as

$$g_1^{*} = \max\left\{ 0, \min\left(1, \frac{S(\alpha C + \beta I))(\lambda_1 + \lambda_2 p) + (1-p)(\alpha C + \beta I)}{C_1} \right) \right\}$$

$$g_2^{*} = \max\left\{ 0, \min\left(1, \frac{(\lambda_2 + \lambda_3 \phi) C}{C_2} \right) \right\}$$

$$g_3^* = \max\left\{0, \min\left(1, \frac{(\lambda_3 - \lambda_4)I}{C_3}\right)\right\}.$$

Proof:

We use the classical finding of [22] to prove the theorem. First, we differentiate the Hamiltonian in (18) with respect to each state variables in order to obtain the set of the adjoint variables. Thus,

$$\frac{d\lambda_1}{dt} = \frac{-\partial H}{\partial S} = -\lambda_1\left(-\mu - (\alpha C + \beta I)(1-g_1)\right) - \lambda_2\left((1-g_1)p(\alpha C + \beta I)\right) - \lambda_3\left((1-g_1)(1-p)(\alpha C + \beta I)\right)$$

$$\frac{d\lambda_2}{dt} = \frac{-\partial H}{\partial C} = -\lambda_1\left(-\alpha S(1-g_1)\right) - \lambda_2\left((1-g_1)p\alpha S - \mu - (\theta + g_2)\right) - \lambda_3\left((1-g_1)(1-p)\alpha S + (1-g_2)\theta\right) - X_1$$

$$\frac{d\lambda_3}{dt} = \frac{-\partial H}{\partial I} = -\lambda_1\left(-\beta S(1-g_1)\right) - \lambda_2\left((1-g_1)p\beta S\right) - \lambda_3\left((1-g_1)(1-p)\beta S - (g_3 + \gamma) - (\mu + \delta)\right)$$
$$- \lambda_4(g_3 + \gamma) - X_2$$

$$\frac{d\lambda_4}{dt} = \frac{-\partial H}{\partial R} = -\lambda_1\varepsilon + \lambda_4(\mu + \varepsilon).$$

Also, to find the optimal control of the control variables sets g_1, g_2, g_3 using partial differential equation

$$\frac{\partial H}{\partial g_i} = 0; i = 1,2,3.$$

For g_1^*, $\frac{\partial H(S,C,I,R,W)}{\partial g_1} = 0$,

$$\therefore g_1 = \frac{S(\alpha C + \beta I)(\lambda_1 + \lambda_2 p) + (1-p)(S\alpha C + \beta I)}{C_1}.$$

For g_2^*, $\frac{\partial H(S,C,I,R,W)}{\partial g_2} = 0$,

$$\therefore g_2 = \frac{(\lambda_2 + \lambda_3\phi)C}{C_2}.$$

For g_3^*, $\dfrac{\partial H(S,C,I,R,W)}{\partial g_3} = \quad 0,$

$\therefore\ g_3 = \dfrac{(\lambda_3 - \lambda_4)I}{C_3}.$

Therefore, $g_1^* = \max\left\{0, \min\left(1, \dfrac{S(\alpha C + \beta I)(\lambda_1 + \lambda_2 p) + (1-p)(S\alpha C + \beta I)}{C_1}\right)\right\},$

$g_2^* = \max\left\{0, \min\left(1, \dfrac{(\lambda_2 + \lambda_3\phi)C}{C_2}\right)\right\}$,

$g_3^* = \max\left\{0, \min\left(1, \dfrac{(\lambda_3 - \lambda_4)I}{C_3}\right)\right\}.$

Based on the prior boundedness and the associated state and adjoint variables, the uniqueness of the optimal control has been established. By standard control arguments which involves the bound on the control, we can say that

$$g_1^* = \begin{cases} 0 & if \quad g_1 \le 0 \\ g_1 & if \quad 0 < g_1 < 1 \\ 1 & if \quad g_1 \ge 1 \end{cases}$$

$$g_2^* = \begin{cases} 0 & if \quad g_2 \le 0 \\ g_2 & if \quad 0 < g_2 < 1, \\ 1 & if \quad g_2 \ge 1 \end{cases} \qquad g_3^* = \begin{cases} 0 & if \quad g_3 \le 0 \\ g_3 & if \quad 0 < g_3 < 1. \\ 1 & if \quad g_3 \ge 1 \end{cases}$$

This completes the proof.

5. RESULTS AND DISCUSSION

5 .1 Numerical simulation of the optimal control

We applied the iterative technique to achieve the optimal solution, taking advantage of the initial conditions of the state system, we used a forward fourth-order Runge-Kutta approach to solve the state equations. Further, due to the final conditions for the adjoint scheme, we used the current

iterations solutions of the state equation to solve the adjoint equations by a backward fourth-order Runge-Kutta method. Each control continues to be updated by integrating its previous and characterization values. The updated controls are used to repeat the solutions. This process goes on until two successive iterations are sufficiently close. The simulation was carried out using the values given in Table 2. We considered the following Initial values for our simulation. $S(0) = 500$, $C(0) = 150$, $I(0) = 200$, $R(0) = 300$. We also used the following values for the coefficients for the state variables and controls X_1 =0.2, X_2, =0.2, C_1, =0.02, C_2, 0.02, and $C_3 = 0.15$.

5.2 Optimal prevention via proper hygiene

For this approach, we used only prevention by proper hygiene g_1 to optimize the objective function *J*, while setting the control on screening g_2 and treatment g_3 to zero. The findings in Figures 2(a) and 2(b) indicate a substantial difference with an optimal strategy in the infected carriers and infected individuals, respectively compared to uncontrolled infected carriers and infected individuals. Figure 2(a) indicates that the control techniques resulted in a dramatic reduction in the population of infected carriers, as opposed to a rise in the uncontrolled scenario. The uncontrolled case also contributed to an increased number of infected individuals in Figure 2(b), however, the control approach contributed to a decrease in the population of individuals infected. Figure 2(c) is the control profile on prevention via proper hygiene g_1. Here we see that the ideal prevention by proper hygiene control g_1 is at the upper limit up to t_f =3 months before falling to the lower limit.

5.3 Optimal screening

With this strategy, we used only screening g_2 to optimize the objective function *J,* while setting the control on prevention by proper hygiene g_1 and treatment g_3 to zero. The results in Figures 3(a) and 3(b) show a considerable difference with an optimal strategy in the infected carriers and infected individuals, respectively compared to uncontrolled infected carriers and infected individuals. Figure 3(a) shows that this strategy has more effect than Figure 3(b) on the infected carrier. This is because screening the infected carriers allows individuals to know their conditions

of health and to go for early treatment. The control profile on screening g_2 is shown in Figure 3(c), which shows that the control g_2 reached the top bound up to $t_f = 3$ months before falling to the lower limit.

5.4 Optimal treatment

For this technique, to optimize the objective function *J*, only treatment g_3 is used as a control, while control on prevention by proper hygiene g_1 and screening g_2 are set to zero. The findings in Figures 4(a) and 4(b) indicate a substantial difference between infected carriers and infected population with control strategy compared to infected carriers and infected individuals without control. Figure 4(a) indicates that the population of infected carrier decreases as against an increase in the uncontrolled situation. The uncontrolled case also led to an increased number of infected persons in Figure 4(b), while the control approach led to a rapid reduction in the number of infected persons as a result of treatment although after one and a half month, the number of infected individuals increases again with lower level compared to non-control case. Figure 4(c) is the control profile on treatment g_3 which shows that the optimal treatment control is at the top bound up to $t_f = 1.5$ months, then reduces slowly to the lower bound

5.5 Optimal prevention via proper hygiene and screening

With this strategy, prevention via proper hygiene g_1 and screening g_2 are used as controls to optimize the objective function *J,* while the control on treatment g_3 is set to zero. The findings in Figure 5(a) and 5(b) show a significant difference in the infected carriers and infected individuals, respectively with optimal control compared to infected carriers and infected individuals without control. Figure 5(a) shows that the combination of the control, prevention through proper hygiene g_1 and screening g_2 resulted in a dramatic reduction in the population of infected carriers as compared to an increase in the uncontrolled case. Further, with controls the number of infected individuals reaches zero after approximately 0.7 months. This combination leads to a decrease in the population of the infected carrier more than the population of the infected

individuals in Figure 5(b). Figure 5(c) is the control profile on prevention via proper hygiene g_1 and screening g_2 receptively. The control g_1 shows that the optimal prevention via proper hygiene is at the upper limit up to t_f =3 months before falling to the lower limit whereas the control g_2 is at the top bound up to t_f = 1.4 months, then reduces slowly to the lower bound.

5.6. Optimal prevention via proper hygiene and treatment

With this strategy, prevention is used as control via proper hygiene g_1 and treatment g_3 to optimize the objective function *J*, while control g_2 is set to zero. The findings in Figure 6(a) and 6(b) indicate a substantial difference with an optimal strategy in the infected carriers and infected population compared to uncontrolled infected carriers and infected population. Figure 6(a) indicates that the combination of prevention by good hygiene and treatment as control measures resulted in a decrease in the population of both infected carriers and infected individuals. The uncontrolled case also contributed to a rise in the number of infected persons in Figure 6(b), while the control approach contributed to a reduction in the population of infected individuals. Figure 6(c) is the control profile on prevention via proper hygiene g_1 and treatment g_3. The control g_1 shows that the optimal prevention through proper hygiene is at the upper limit up to 2.9 months before falling to the lower limit while the control on g_3 is at the upper limit up to 1.1 month, then reduces slowly to the lower bound.

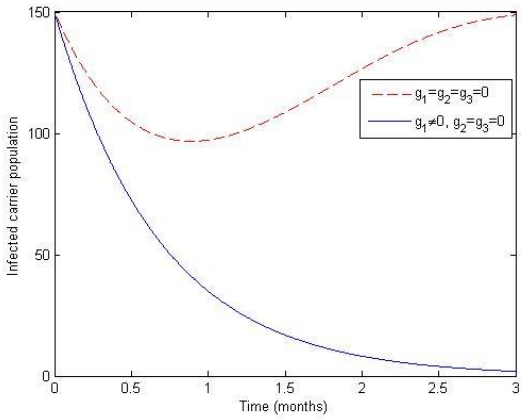
5.7 Optimal screening and treatment

Screening g_2 and treatment g_3 are used as control to optimize the objective function *J,* while setting the control prevention by proper hygiene g_1 to zero. The findings in Figure 7(a) and 7(b) show a significant difference between infected carriers and infected persons with an optimal strategy compared to infected carriers and uncontrolled persons. Figure 7(a) indicates that the combination of screening and treatment as control measures is very successful for the infected carriers, resulting in a significant reduction in the number of infected carriers and reaches zero after 0.7 month as compared to a rise in the uncontrolled case. This combination also has more

effect than those of people infected. The uncontrolled case also contributed to a rise in the number of infected population in Figure 7(b), while the control approach contributed to a reduction in the number of infected population. Figure 7(c) is the control profile on screening g_2 and treatment g_3. The control g_2 is at the upper limit up to t_f =2.9 months before falling to the lower limit. Meanwhile, the control g_3 is at the top bound up to t_f = 1.1 months then reduces slowly to the lower bound.

5.8 Optimal prevention via proper hygiene, screening and treatment

For this approach, all controls, prevention by proper hygiene g_1, screening g_2 and treatment g_3 were applied to maximize objective function *J.* The results in Figure 8(a) and 8(b) show a significant difference with an optimal strategy in the infected carriers and infected individuals, respectively compared to uncontrolled infected carriers and infected individuals. Figure 8(c) is the control profile on prevention by proper hygiene g_1, screening g_2 and treatment g_3.The control g_1 shows that the optimal prevention by proper hygiene is at the upper limit up to t_f =3 months before falling to the lower limit, while the control g_2 is at the upper limit up to t_f =2.9 months before falling to the lower limit. On the other hand, the control g_3 is at upper bound t_f =1.1 months before falling to the lower limit. We conclude that, the combination of all three controls results in a stronger reduction in population of infected carriers and infected individuals. However, the population of infected individuals reaches zero after approximately half month, which gives the best results compared to the previous 6 strategies mentioned above. Therefore, in a given period, implementing this strategy is successful in eradicating the disease epidemic eventually.



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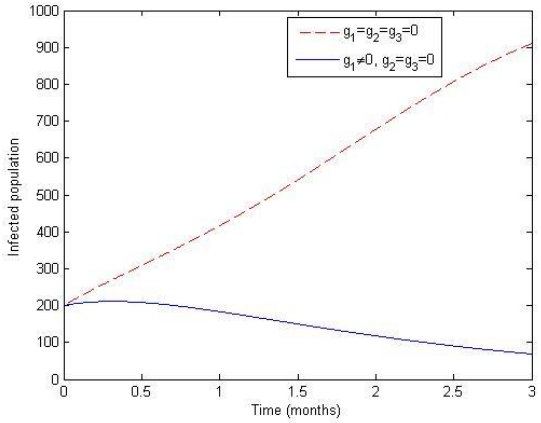

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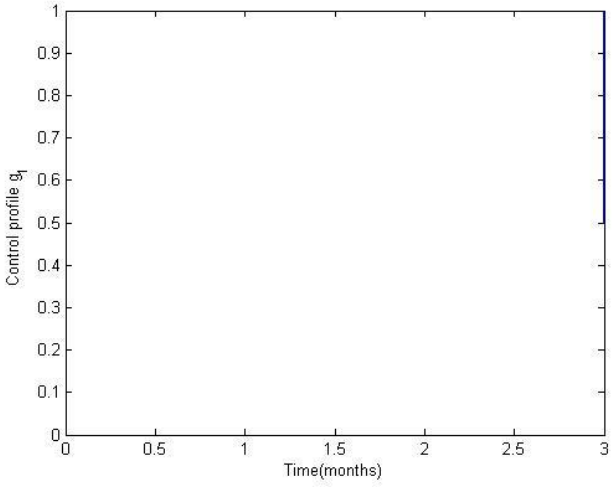
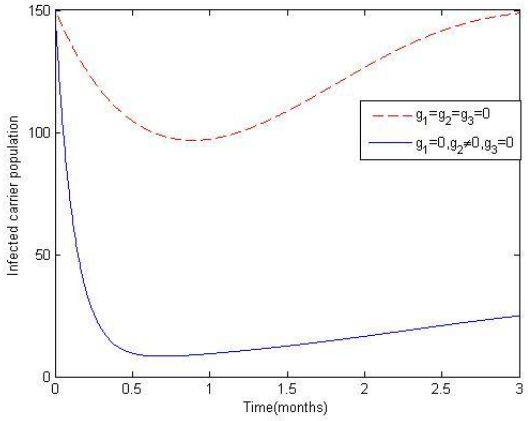


(c)

FIGURE 2**.** Simulations showing the effects of prevention via proper hygiene on infected carrier and infected individuals.



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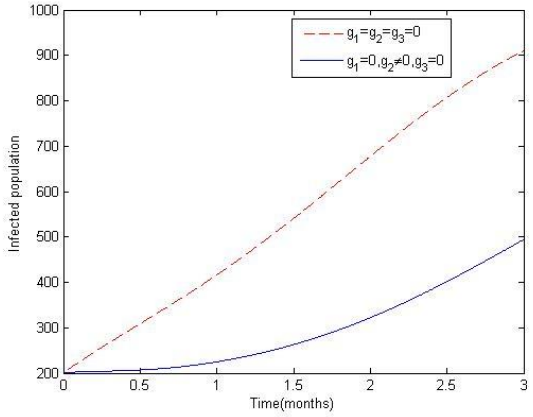

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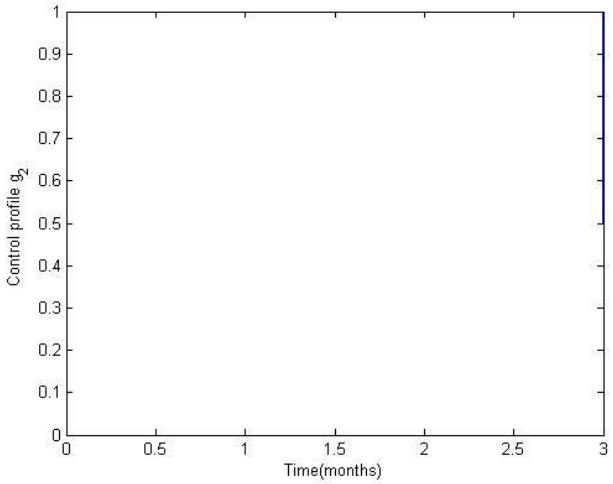
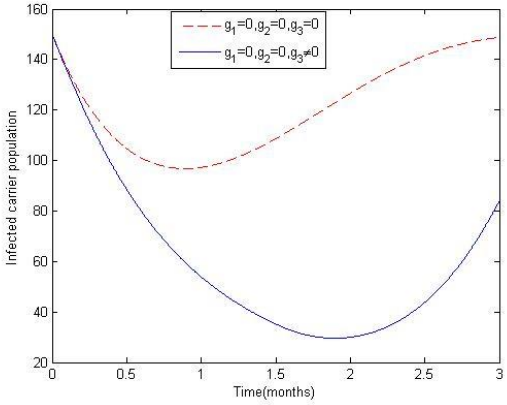


(c)

FIGURE 3. Simulations showing the effects of screening on infected carrier and infected individuals.



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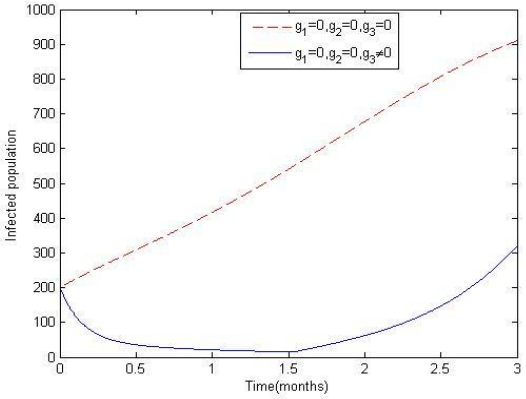

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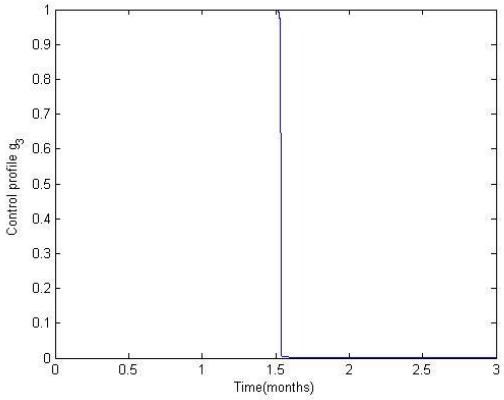
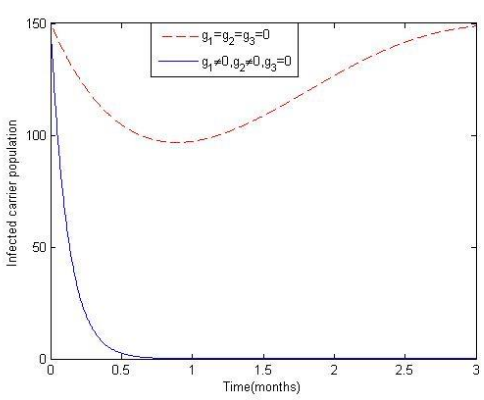


(c)

FIGURE 4. Simulations showing the effects of treatment on infected carrier and infected individuals.



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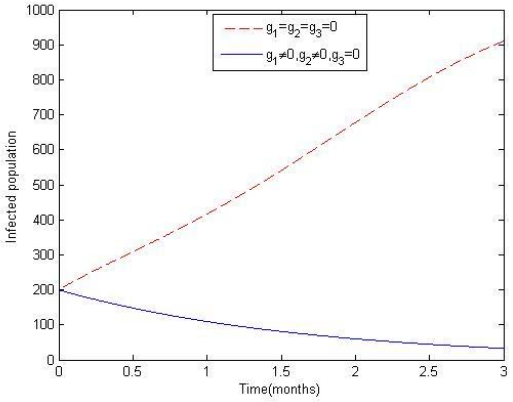

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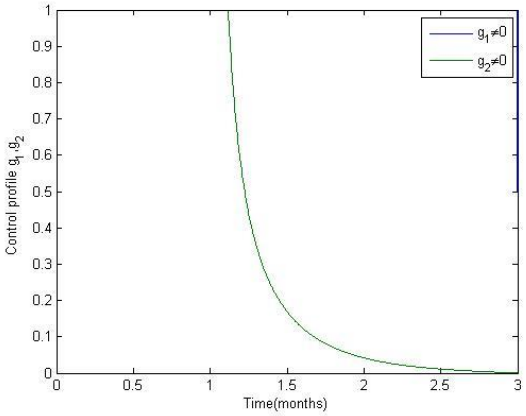
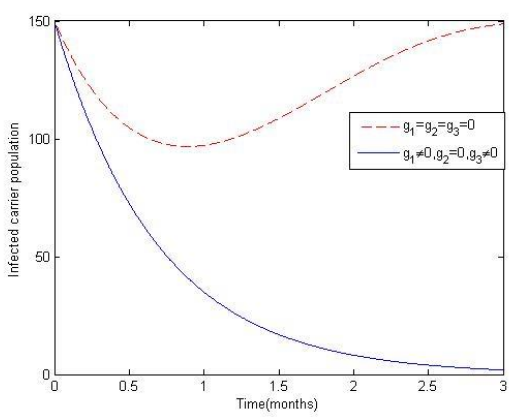


(c)

FIGURE 5**.** Simulations showing the effects of prevention via proper hygiene and screening on infected carrier and infected individuals.



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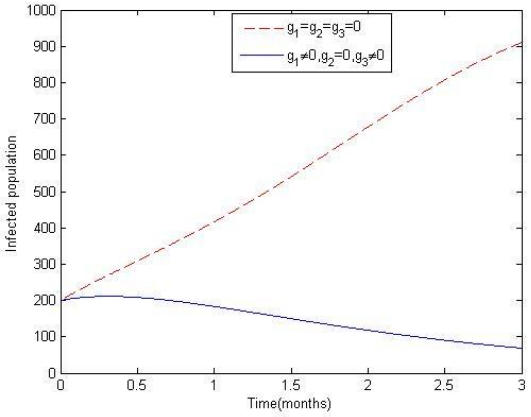

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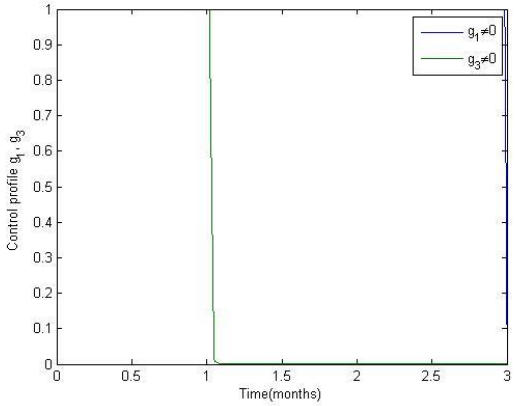
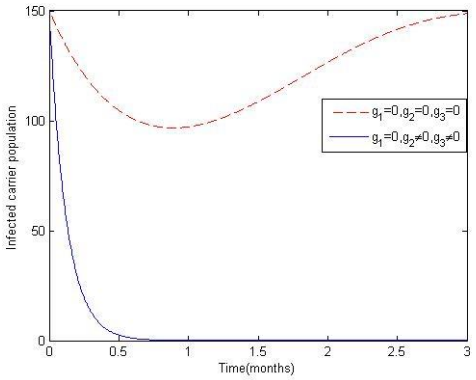


(c)

FIGURE 6. Simulations showing the effects of prevention via proper hygiene and treatment of infected carrier and infected individuals.



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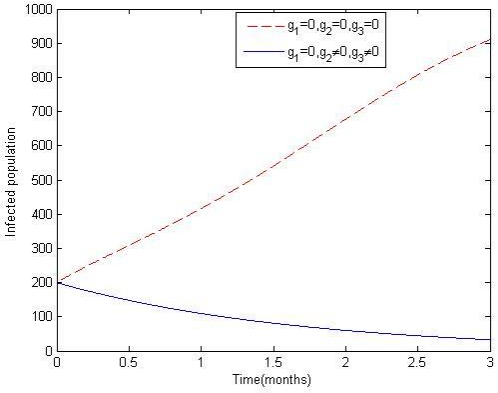

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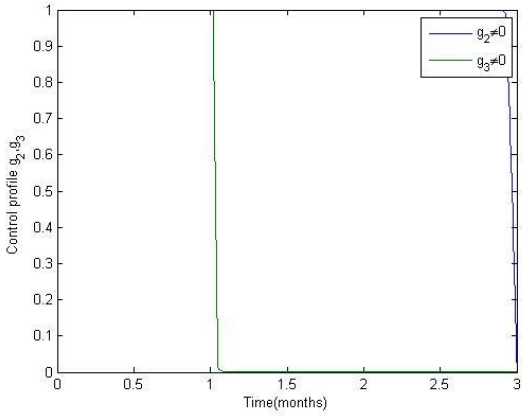
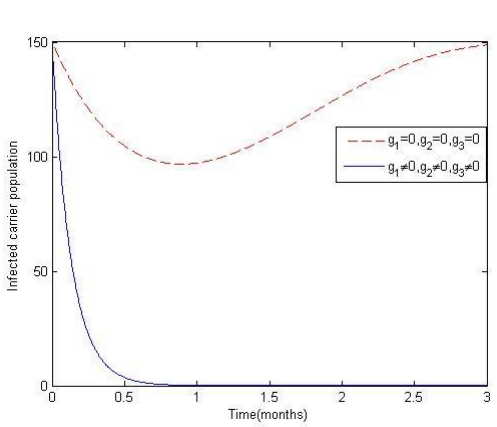


(c)

FIGURE 7. Simulations showing the effects of screening and treatment on infected carrier and infected individuals.



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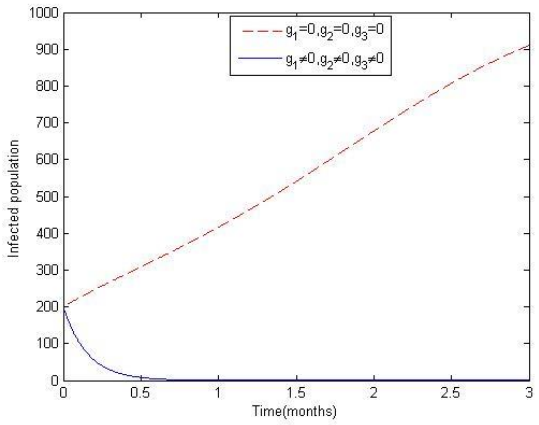

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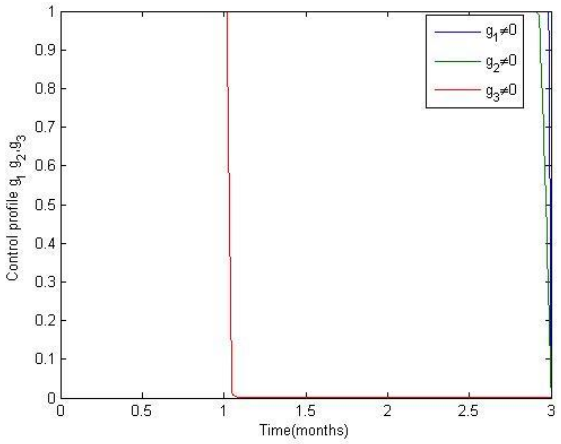

(c)

FIGURE 8. Simulations showing the effects of prevention via proper hygiene, screening and treatment on infected carrier and infected individuals.

6. CONCLUSION

In this study, a compartmental model of *SCIR* governed by a system of nonlinear differential equations is proposed. We have demonstrated the boundary of solutions and shown that all solutions are non-negative. Two equilibrium points (disease-free and endemic) are obtained and their stability depends on the basic reproduction number. When the basic reproduction number is less than unity, the disease is eventually eradicated and the disease-free equilibrium is globally stable. On the contrary, the disease persists and the endemic equilibrium point is globally stable when it satisfies some condition i.e. $\bar{b}>0$. Our sensitivity analysis suggests that we

should try to reduce the transmission rate and increase the transferring from carrier individuals to infected individuals to prevent greater contact. Hence, we further extend the model in (1) to optimal control problems by adding three control variables which are prevention via proper hygiene, screening of the infected carriers which enable them to know their health conditions and to go for early treatment and treatment of the infected individuals. We performed numerical simulations in 7 different strategies of different controls. Our results show that using combinations of all three controls gives the best result in reducing the spread of disease overall. Therefore, we should encourage these three controls in order to control the spread of infectious disease.

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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