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Stability Analysis and Optimal Control of Vaccination and Treatment of a *SIR* Epidemiological Deterministic Model with Relapse

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Abstract. In this paper, we studied and formulated the relapsed *SIR* model of a constant size population with standard incidence rate. Also, the optimal control problem with treatment and vaccination as controls, subject to the model is formulated. The analysis carried out on the model, clearly showed that the infection free steady state is globally asymptotically stable if the basic reproduction number is less than unity, and the endemic steady state, also, is globally asymptotically stable if the basic reproduction number (R_0) is greater than unity. The results obtained from the simulations were analyzed and discussed.

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1. Introduction

In some infectious diseases, the relapse phenomenon occurs when a previously infected host becomes infected again due to subsequent relapse or reactivation of the disease. Some diseases like bovine tuberculosis, human herpes virus, malaria, e.t.c., can be modeled in a similar manner like the model considered in this paper due

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to the disease's ability to reactivate itself by making their host become infectious again. If adequate and timely interventions like vaccination, treatment, educational enlightenment, campaign e.t.c., are put in place within the host community, then, such diseases with high endemicity would be eradicated.

Mathematical epidemiological models have been employed as an important tool to analyze the control and the spread of diseases. Reproduction number, (R_0), being a key factor in epidemiological studies, have been studied by several authors, in which [2, 5, 6, 11, 13, 14] have proved very useful in this study. One of the first epidemic models with relapse was studied and formulated by [3] in which the relapse factor was incorporated into the model. Later, [10] formulated a model in a constant population with latency and relapse. Also the work of [1, 4, 19, 20, 21, 22, 23, 24] have proved very useful in studying both the local and global properties of general *SIR* and *SIRS* epidemic models with non - linear transmission rate.

Optimal control theory have been another important aspect of mathematics employed to control the spread of disease and a decision tool involving epidemiological situations when control measures such as treatment, vaccine, e.t.c are available. [7] used optimal control theory to determine the condition of the elimination of tumor cells in an individual under treatment for cancer. The literature of [9, 12, 8, 9, 16, 17, 18] have been useful in the application of optimal control process to mathematical epidemiological models.

In this paper, we worked on how the two controls, vaccination (μ_1) and treatment (μ_2) strategies can be combined optimally, so that the cost of implementing it would be reduced and at the same time the disease would be eradicated within a period of time. Our work slightly differs from the other cited literatures, because we considered and studied the stability conditions of the *SIR* model which incorporates relapse with standard incidence rate and a constant population size. While, the objective functional reduces the numbers of infective at the control period to a level in which the disease would be eradicated and die out.

The rest of the paper is organized as follows. In section 2, the model is formulated and analyzed. Section 3 presents the basic reproduction number being studied and obtained. Also, in section 4, the two equilibria at disease free and endemic is obtained, while the local and global stability analysis is investigated at the equilibria. Section 5, the optimal control is characterized and the optimality of the systems are derived using the Pontryagin maximum principle. Also, the numerical simulations were performed, and the results obtained is concluded analytically.

2. Mathematical model formulation

In this paper, the compartmental models are subdivided into disjointed mutually exclusive classes. The total host population breaks the total host population into different subgroups that are the susceptible class S, Infectious class I and individuals that are infected but recovered or removed R. In the *SIR* model, the relapse parameter σ is incorporated into the model thereby making the recovered individual to be susceptible to the disease by the reactivation of the disease. The model is presented as follows

$$\begin{aligned}
\dot{S} &= \mu N - \frac{\beta SI}{N} - \mu S - u_1 S + \sigma R,\\
\dot{I} &= \frac{\beta SI}{N} - \mu I - u_2 I + \alpha R,\\
\dot{R} &= u_2 I - (\mu + \alpha + \sigma) R,
\end{aligned} \tag{1}$$

subject to initial conditions $S(t) > 0$, $I(t) > 0$, $R(t) \geqslant 0$.

In (1), all the parameters in the model are all positive constants. The parameter μN, is the recruitment rate of the susceptible class. While, μ is the natural death rate of the population , β is the disease transmission rate, u_1 is the proportion of the susceptible that are vaccinated per unit time, u_2 is the proportion of the infectives that is treated per unit time, α is the disease induced death rate.
The total population,

$$N = S + I + R \tag{2}$$

where,

$$R = N - S - I \tag{3}$$

substituting (3) into (1) gives,

$$\begin{aligned} \dot{S} &= \mu N - \frac{\beta SI}{N} - \mu S - u_1 S + \sigma(N - S - I) \\ \dot{I} &= \frac{\beta SI}{N} - \mu I - u_2 I + \alpha(N - S - I) \end{aligned} \tag{4}$$

Non - dimensionalising (4), let $\dot{S} = \frac{s}{N}$and $\dot{I} = \frac{i}{N}$

$$\begin{aligned} \dot{S}N &= \mu N - \beta siN - \mu SN - u_1 SN + \sigma(N - sN - iN) \\ \dot{I}N &= \beta siN - \mu iN - u_2 iN + \alpha(N - sN - iN) \end{aligned} \tag{5}$$

dividing (5) through by N, and solving further yeilds,

$$\begin{aligned} \dot{S} &= \mu - \beta si - (\mu + u_1)s + \sigma(1 - s - i) \\ \dot{I} &= \beta si - \mu I - u_2 I + \alpha(1 - s - i) \end{aligned} \tag{6}$$

2.1 *Analysis of the model*

To obtain the invariant region of (1), we differentiate both sides of (2) to obtain

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \tag{7}$$

also, adding up (1) becomes,

$$\dot{N} = \mu N(t) - \mu \tag{8}$$

integrating both sides of (8) becomes;

$$\int \frac{dN}{dt} = \int \mu N(t) - \mu \tag{9}$$

such that,

$$\frac{-1}{\mu} ln(\mu - \mu N) \leqslant t + c \tag{10}$$

becomes,

$$N = \frac{N}{\mu} + Ce^{-\mu t} \tag{11}$$

where C is a constant. Therefore,

$$lim_{t\longrightarrow 0}\Big(\frac{N}{\mu} + \frac{C}{e^{\mu t}}\Big) = \frac{N}{\mu} \tag{12}$$

this implies that,

$$S \leqslant \frac{N}{\mu} + \frac{C}{e^{\mu t}}, I \leqslant \frac{N}{\mu} + \frac{C}{e^{\mu t}}, R \leqslant \frac{N}{\mu} + \frac{C}{e^{\mu t}} \tag{13}$$

since all the sum is equal to N, therefore the upper bound of (1) is $\frac{N}{\mu} + \frac{C}{e^{\mu t}}$, and the lower bound is 0. It clearly shows that, all the solutions of, $S(t), I(t), R(t)$ of (1) are bounded. Therefore,

$$\xi = \Big[x = (S, I, N) \in \Re^{+3} | S \geqslant 0, I \geqslant 0, S + I \leqslant N \leqslant \frac{N}{\mu}\Big] \tag{14}$$

is positively invariant.
Hence, for the initial starting point $x \in \Re^{+3}$, the trajectory remains in ξ which makes the model system (1) to be epidemiologically realistic and mathematically well posed.

3. Reproduction number (R_0)

In (1), μN is the recruitment rate and μ is the per capita natural death rate, which are assumed to be constant. According to Castillo - Chavez and Zilhan Feng [23] that, let,

$$X = (S, R), Z = I, U_0 = \Big(\frac{N}{\mu}, 0, 0\Big) \tag{15}$$

also,

$$\mu N = \beta - (\mu + u_2 + \alpha) \tag{16}$$

where $M = \beta$ and $D = (\mu + u_2 + \alpha)$. β is the average number of susceptible individuals infected by one infectious individual per unit time and $\frac{1}{\mu+u_2+\alpha}$ is the mean length of infectious period. Then,

$$R_0 = MD^{-1} = \frac{\beta}{\mu + u_2 + \alpha} \tag{17}$$

R_0 is the number of secondary infectious individual introduced into the population of susceptible during individual's infection per unit time.
if $R_0 > 1$, an outbreak of the disease occurs. If $R_0 < 1$, then the disease will be eradicated.

4. Existence of the steady states

The existence of the steady state solutions of the model is investigated to know what will likely happen to the disease in a short or long term in the host population if, the disease would be eradicated or it will persist and become endemic.

4.1 *Disease free equilibrium*

Assuming that there is no infection in the system (4), i.e.$(i = 0)$, setting,

$$S = \mu - \beta si - (\mu + u_1)s + \sigma(1 - s - i) = 0 \tag{18}$$

and,

$$I = \beta si - (\mu + u_2)i + \alpha(1 - s - i) = 0 \tag{19}$$

becomes,

$$\alpha(1 - s) = 0 \tag{20}$$

if $\alpha \neq 0, s = 1$, then, the equilibrium points, $(s, i) = (1, 0)$ is the disease free equilibrium.

4.2 *Local stability analysis of the disease free equilibrium*

To check for the local stability of the disease free equilibrium of the model, we obtained the jacobian variational matrix of the systems of the given equations. Let,

$$\begin{aligned} f_1 &= \mu - \beta si - (\mu + u_1)s + \sigma(1 - s - i) \\ f_2 &= \beta si - (\mu + u_2)s + \alpha(1 - s - i) \end{aligned} \tag{21}$$

so that,

$$J|(s, i) - \lambda| = \begin{pmatrix} \frac{\partial f_1}{\partial s} & \frac{\partial f_1}{\partial i} \\ \frac{\partial f_2}{\partial s} & \frac{\partial f_2}{\partial i} \end{pmatrix} \tag{22}$$

at disease free equilibrium points becomes,

$$\begin{pmatrix} -\beta i - (\mu + u_1) - \lambda & -\beta s - \sigma \\ \beta i - \alpha & \beta s - (\mu + u_2) - \alpha - \lambda \end{pmatrix} \tag{23}$$

solving the determinant in (18) gives,

$$\lambda^2 + \lambda[(\mu + u_1 + \sigma - \beta + (\mu + u_2 + \alpha)] - (\mu + u_1 + \sigma)(\beta - (\mu + u_2 + \alpha)] \tag{24}$$

if,

$$\beta > (\mu + u_2 + \alpha) \Leftrightarrow [\beta(\mu + u_2 + \alpha)] > 0 \tag{25}$$

also,

$$\frac{\beta}{\mu+u_2+\alpha} > \frac{\mu+u_2+\alpha}{\mu+u_2+\alpha} = \Big(\frac{\beta}{\mu+u_2+\alpha} - 1\Big) > 0 \tag{26}$$

where $(R_0 - 1) > 0$ implies that $R_0 < 1$, shows that the disease free equilibrium points of the model (6) is locally asymptotically stable.

4.3 *Global stability of the disease free equilibrium*

To prove the global stability of the disease free equilibrium points in ξ for $R_0 \leqslant 1$, we define a Lyapunov function that if, $V : \xi \longrightarrow R$ then, $V(s, i) = i$

$$V(s,i) = \frac{di}{dt} = [(\beta s - (\mu + u_2) - \alpha)i + \alpha(1 - s)] \tag{27}$$

implies that,

$$\dot{V}(s,i) = (\mu+u_2+\alpha)\Big(R_0 s - \frac{1}{\mu+u_2+\alpha}\Big)i \leqslant 0 \Rightarrow R_0 \leqslant 1 \tag{28}$$

so that,

$$\dot{V}(s,i) = (\mu+u_2+\alpha)\Big(R_0 s - \frac{1}{\mu+u_2+\alpha}\Big)i \leqslant 0 \Rightarrow R_0 \leqslant 1 \tag{29}$$

then, it clearly shows that for $R_0 > 1$, $\dot{V} = 0$ implies that $i = 0$. also, if $R_0 = 1$, $\dot{V} = 0$ implies that $s = 1$. From the La - Salle Lyapunov theorem [6] that, if $s = 1$ and $i = 0$, then, $\frac{di}{dt} = 0$, so that the only positively invariant subset of the $i = 0$, is the disease free equilibrium point, which is globally stable in the interior of ξ for $R_0 \leqslant 1$

4.4 *Endemic equilibrium*

Assuming that the infection persists in the system (4), such that

$$(s^*, i^*) = (i \neq 0) \tag{30}$$

, then, the equilibrium points

$$(s^*, i^*) = \Big(\frac{\mu+\sigma(1-i)}{\beta i+\mu-\sigma+u_1}, -\frac{\alpha(-1+s^*)}{\beta s^* - (\mu+u_2)+\alpha}\Big) \tag{31}$$

is the endemic equilibrium point.

4.5 *Local stability of the endemic equilibrium*

To obtain the local stability of the endemic equilibrium points, we set the right hand side of (6) to zero such that,

$$\begin{aligned} f_1 &= \mu - \beta si - (\mu + u_i)s + \sigma(1 - s - i) \\ f_2 &= \beta si - (\mu + u_2)i + \alpha(1 - s - i) \end{aligned} \tag{32}$$

and the Jacobian of the variational matrix is obtained from (28) as,

$$J|(s,i)-\lambda| = \begin{pmatrix} \frac{\partial f_1}{\partial s} & \frac{\partial f_1}{\partial i} \\ \frac{\partial f_2}{\partial s} & \frac{\partial f_2}{\partial i} \end{pmatrix} \tag{33}$$

where (33) becomes,

$$|J-\lambda I| = \begin{pmatrix} -\beta i^* - (\mu+u_1) - \lambda & -\beta s^* - \sigma \\ \beta i^* - \alpha & \beta s^* - (\mu+u_2) - \alpha - \lambda \end{pmatrix} \tag{34}$$

The characteristics polynomial of the variational matrix (34) at the endemic equilibrium points, $E^*(s^*, i^*)$ is,

$$\lambda^2 + a_1\lambda + a_2 \tag{35}$$

where,

$$a_1 = (2\mu + \alpha + u_1 + u_2) \tag{36}$$

and,

$$a_2 = \beta s^*(\mu+\alpha+\lambda+u_1) + \beta i^*(\mu+\alpha+\sigma+\lambda+u_2) + (\alpha+u_1+u_2)\mu + (\sigma+u_1)\alpha + u_1u_2. \tag{37}$$

The Routh - Hurwitz criteria states that the variational matrix have negative real parts if and only if $a_1 > 0$ and $a_2 > 0$. Also, the trace of (34) is given as,

$$-2\mu - u_1 - u_2 + \sigma - \alpha + \frac{\beta\alpha(-1+s^*)}{\beta s^* - \mu - u_2 + \alpha} - \frac{\beta[\mu + \sigma(-1+i^*)]}{\beta s^* + \mu - \sigma + u_1} \tag{38}$$

such that,

$$-2\mu - u_1 + (R_0 - 1) \tag{39}$$

which clearly shows that the trace is negative provided that $R_0 > 1$. Also, the determinant of (34) is given as,

$$(\beta i^* - \alpha)(\beta s^* - \sigma) - [\beta i^* - (\mu + u_1)][R_0 - 1] \tag{40}$$

implies that the determinant of the matrix is positive provided that $R_0 > 1$. Thus, the endemic equilibrium $E^* = (s^*, i^*)$ is locally asymptotically stable.

4.6 *Global stability of the endemic equilibrium*

The global stability of the endemic equilibrium of (31)is solved via the direct method of Lyapunov and La - Salle invariance principle. We consider the non linear Lyapunov functions.

$$V : \xi_+ \longrightarrow R, \xi_+ = [(s,i,n) \in \xi_+, s > 0, i > 0, n > 0] \tag{41}$$

such that,

$$V(s,i,n) = \frac{(n-n^*)^2}{2} + \frac{(s-s^*)^2}{2} + \frac{\epsilon}{ki}\Big[i - i^* - i^* ln\Big(ln\frac{i}{i^*}\Big)\Big] \tag{42}$$

differentiating (42) to obtain,

$$\dot{V}(s,i,n) = (n-n^*)\dot{n} + (s-s^*)\dot{s} + \frac{\epsilon}{kl}\Big(1 - \frac{i^*}{i}\Big)i \tag{43}$$

so that,

$$\dot{V} = (n-n^*)(\mu n^* - \mu - u_i s^*) + (s-s^*)(\mu n - \beta si - (\mu+u_1)s + \sigma(1-s-i) + \frac{\epsilon}{ki}\Big(i - \frac{i^{*2}}{i}\Big)(\beta si - (\mu+u_1)i + \alpha(1-s-i) \tag{44}$$

from (6),

$$un = \beta s^* i^* - (\mu+u_1)s^* + \sigma(1-s^*-i^*) = \mu n^* - \mu - u_1 s^* \tag{45}$$

and,

$$\beta s^* i^* = (\mu+u_2)i^* + \alpha(1-s^*-i^*) = \beta s^* i^* - (\mu+u_1)s^* + \sigma(1-s^*-i^*) \tag{46}$$

so that,

$$\dot{V} = (n-n^*)(\mu n^* - \mu - u_1 s^*) + (s-s^*)(\mu n - \beta si - (\mu+u_1)s^* + \sigma(1-s-i) + \frac{\epsilon}{kl}\Big(i - \frac{i^*2}{i}\Big)(\beta si - (\mu+u_1)i + \alpha(1-s-i)). \tag{47}$$

also,

$$\dot{V} = (n-n^*)(\mu n^* - \mu - u_1 s^* - \mu - u_1 s^* - \mu - u_1 s^*) + (s-s^*)(\beta s^* i^* - (\mu+u_1)s^* + \sigma(1-s^*-i^*)) - \beta si - (\mu+u_1)s + \sigma(1-s-i) + \frac{\epsilon}{k}(i-i^*)(\beta si - \beta s^* i) \tag{48}$$

so that,

$$\dot{V} = \mu(n-n^*)^2 + u_1(n-n^*)(i-i^*) - (\mu+u_1)(s-s^*)^2 + \beta(s-s^*)(s^* i^* - si) + \sigma(s-s^*)(i-i^*) + \frac{\beta\epsilon}{k}(i-i^*)(s-s^*) \tag{49}$$

Hence, $\dot{V} = 0$ if and only if $s = s^* = \frac{\epsilon}{k}, i = i^* = 0$ and $n = n^*$, by the La - Salle's invariant principle [6], the largest compact invariant set in $(s,i) \in \xi : \dot{V} = 0$ is the singleton (E^*). (E^*) is the endemic equilibrium. Thus, the endemic equilibrium is globally asymptotically stable in the region ξ.

5. Optimal control problem

In this section, we propose and analyze an optimal control problem applied to the disease model dynamics governed by (6). We included a control function $u(*)$ to the model which represents the fraction of infected individual i that are subjected to treatment and vaccination until recovery. It is however expected that the control takes value in the closed set $[0, 1]$, where $u = 0$ means, no control measure and $u = 1$ means that all infected individual are subjected to vaccination and treatment. We define the objective functional as,

$$Z = min_{u_1 u_2} i(T) + \frac{1}{2}\int_0^T (W_1 u_1^2 + W_2 u_2^2)dt \tag{50}$$

subject to the system (6) with states initial conditions. Also, the control set Ω is Lebesgue measurable and thus defined as,

$$\Omega = \big[(u_1(t), u_2(t))|0 \leqslant u_1 \leqslant u_1 max \leqslant 1, 0 \leqslant u_2 \leqslant u_2 max \leqslant 1, t \in [0, T]\big] \tag{51}$$

Also, W_1 and W_2 are the relative weights attached to the cost of treatment and vaccination. u_1 is the control function for the proportion of susceptible individuals subjected to vaccination per unit time and u_2 is the control function for the proportion of infected individuals subjected to treatment per unit time. Where u_1 and u_2 takes value between 0 and 1. In addition, u_{1max} and u_{2max} respectively depend on the available resources to implement each of the control measures. Furthermore, $W_1 u_1^2$ and $W_2 u_2^2$ denote the cost associated with vaccination and treatment which includes, vaccine cost, vaccine storage cost, drug cost, medical test and diagnosis costs e.t.c. The cost take a non - linear form, and $i(t)$ is the terminal cost which the goal is to minimize the proportion of the infective individual after the implementation of the control program.

5.1 *Characterization of the optimal control pairs*

The control pairs (u_1^*, u_2^*) will be characterized to accomplish the set objectives and the states (s^*, i^*). According to the Pontryagin maximum principle [9] with a fixed final time (T). Then, there exists a non trivial absolute continuous mapping:

$$\lambda : [0, T] \to R^{+2}, \lambda(t) = [\lambda_1(t), \lambda_2(t)] \tag{52}$$

is called the adjoint vector, such that, the control system,

$$s^{'} = \frac{\partial H}{\partial \lambda_1}, i^{'} = \frac{\partial H}{\partial \lambda_2} \tag{53}$$

the adjoint system,

$$\lambda^{'} = \frac{\partial H}{\partial s}, \lambda_2^{'} = -\frac{\partial H}{\partial i} \tag{54}$$

is subject to the transversality condition

$$\lambda_1(T) = 0, \lambda_2(T) = 1 \tag{55}$$

then, the optimal control pairs (u_1^*, u_2^*) is thus given as,

$$u_1^* = min\Big[max\Big(0, \frac{\lambda_1 S}{W_1}\Big), u_1 max\Big], u_2^* = min\Big[max\Big(0, \frac{\lambda_2 S}{W_2}\Big), u_2 max\Big] \tag{56}$$

and

$$H = \frac{1}{2}W_1 u_1^2 + W_2 u_2^2 + \lambda_1(\mu - \beta si - (\mu + u_1)s + \sigma(1 - s - i) + \lambda_2(\beta si - (\mu + u_2)i + \alpha(1 - s - i) \tag{57}$$

is called the Hamiltonian.
we now proceed to differentiate H with respect to u_1 and u_2 by employing the Pontryagin maximum principle [9], where the Hamiltonian is maximized with respect to the optimal control pairs, Then,

$$\frac{\partial H}{\partial u_1} = W_1 u_1 - \lambda_1 s = 0 \tag{58}$$

and

$$\frac{\partial H}{\partial u_2} = W_2 u_2 - \lambda_2 i = 0 \tag{59}$$

also, by substituting $u_1 = u_1^*$ and $u_2 = u_2^*$ and solving for the optimal control pairs we obtain,

$$u_1^* = \frac{\lambda_1 s}{W_1}, u_2^* = \frac{\lambda_2 i}{W_2} \tag{60}$$

which in turn gives the optimality of the system as

$$\begin{aligned} \dot{S} &= \mu - \beta si - (\mu + u_1)s + \sigma(1 - s - i) = 0, \\ \dot{I} &= \beta si - (\mu + u_2)i + \alpha(1 - s - i) = 0, \end{aligned} \tag{61}$$

subject to

$$S(0) = S_0, I_{(0)} = I_0, \tag{62}$$

$$\begin{aligned} \dot{\lambda_1} &= \lambda_1(\beta i + \mu + u_1^* + \sigma) - \lambda_2 i, \\ \dot{\lambda_2} &= \lambda_1(\beta s - \lambda_2 i(\beta s - u_2^* - d - \alpha), \end{aligned} \tag{63}$$

subject to

$$\lambda_1(T) = 0, \lambda_2(T) = 0. \tag{64}$$

6. Numerical simulations and discussion of results

Numerical solutions of the proposed model (6) was obtained using Maple ODE solver with different initial starts and parameters in the models as follows;
$\mu = 0.01, \beta = 0.5, \sigma = 0.02, \alpha = 0.03$, it is however pertinent to note that the parameters were incorporated into the model so that the population $N(t)$ will not go extinct. The results obtained were displayed in the Figures 1, 2, 3, 4 and 5 below.

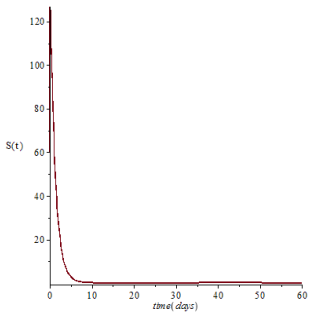

Figure 1. Plot of S(t) against time (t)(days).

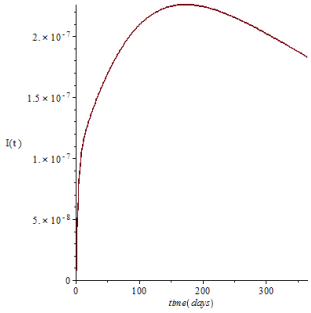

Figure 2. Plot of I(t) against time (t)(days).

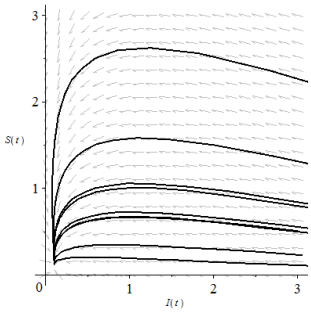

Figure 3. Plot of the phase portrait of $s(t)$ against $i(t)$.

6.1 *Discussion of results*

Figure 1 shows the class of susceptible individuals. It is observed from Figure 1 that Without any control measure in place in which, within a short period of time, there would be a large and quick inflow from the susceptible to the infected class. Therefore control measures should be put in place to reduce contact between the

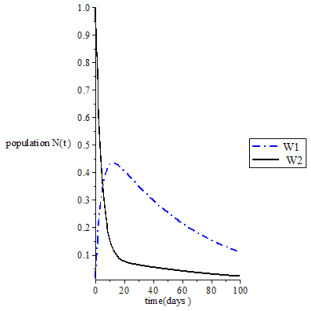

Figure 4. Plot of the population of $N(t)$ for W_1 and W_2 against time(days).

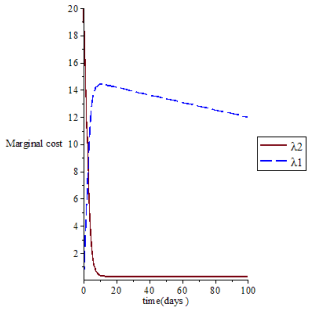

Figure 5. Plot of marginal cost λ_1, λ_2 for W_1 and W_2 against time(days).

susceptible and the infected class. Figure 2 depicts that there is a sharp increase in the level of infectiousness if infective individuals continue to infect people in the host population, and without control measures, the persists and can lead to death in the host population. Figure 3 is the phase portrait showing the movement of the susceptible with the infected class. Due to the incidence rate, the flow of individuals out of the susceptible class is directly proportional to the rate at which people are infected with the disease. The trajectories of the two solutions of $s(t)$ and $i(t)$ are stable and non periodic. Figure 4 shows the profile of the control functions u_1 and u_2 with weights W_1 and W_2 respectively. If the treatment control is applied, it does not really have much effect in bringing down the numbers of infected individuals which peak less than the former. But, if the two controls have the same weight, there might not be a significant difference. Figure 5 clearly shows that after the intersection between the two costs, λ_1 and λ_2 for W_1 and W_2. The marginal cost of treatment peaks higher with time. While, the marginal cost vaccination begins to drop with time, Because, it is more economical to expand the treatment coverage unless it is otherwise unavoidable.

7. Conclusion

In this paper, a deterministic SIR model with standard incidence rate, incorporating relapse and several other parameters is proposed and studied. The SIR model considered in this paper slightly differs from the general SIR and $SEIR$ model worked upon qualitatively and quantitatively, thereby showing that the model is positive, epidemiologically realistic and mathematically non trivial. The steady states at infection free and present is obtained and their local and global stability conditions is investigated. However, the basic reproduction number R_0 shows that if $R_0 < 1$, the disease will be eradicated and if $R_0 > 1$, then , the disease lingers on. The optimal analysis is investigated to show how the optimal combination of treatment and vaccination strategies will completely eradicate the disease with cure

and vaccine within a specified period of time. The Pontryagin maximum principle [9] is employed to characterize the two controls and derive the system optimally. Furthermore, the numerical simulation of the disease profile to the susceptible and infectious class is plotted, firstly, in the absence of control and later on, optimally in the presence of controls u_1 and u_2 to show it is more expensive to treat than to vaccinate in order to put the disease below a certain threshold. Finally, this work can be modeled and extended upon in a similar manner for diseases like malaria, typhoid, tuberculosis, e.t.c. Also, the results obtained from this work can contribute to, and be improved upon when problems arises in epidemiological studies.

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