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Optimal and Global Analysis of the Transmission Dynamics of a SIS-VS Epidemic Model with Non-Linear Incidence Rate

Ogunmiloro OM*

Department of Mathematics, Ekiti - State University, Ado - Ekiti, Ekiti -State, Nigeria

***Corresponding author:** Ogunmiloro OM, Department of Mathematics, Ekiti - State University, Ado - Ekiti, Ekiti -State, Nigeria, Tel: +2348037728658, E-mail: oluwatayo.ogunmiloro@eksu.edu.ng

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Abstract

This paper presents a *SIS-VS* epidemic model of a variable population size of constant recruitment with non - linear incidence rate. Two control variables which are, the media coverage and treatment is applied to control the susceptible, vaccinated and the infected individuals by minimizing the total number of infected individual and the cost associated with it on [0, 1]. The model possess two equilibrium points namely, the disease free and endemic equilibrium. From the derivation of the basic reproduction number (R_0), it is observed that if $R_0 < 1$ the disease free equilibrium is locally and globally stable. Also, if $R_0 > 1$ the endemic equilibrium is both locally and globally stable.

Keywords: Local Stability; Global Stability; Reproduction Number; Optimality System; Pontryagin Maximum Principle

Introduction

In epidemiology, the understanding of the transmission dynamics of compartmental models of emerging and reemerging disease is based on optimizing the use of limited resources in curtailing the full blown spread of the disease in the event of an epidemic breakout. Mathematical model as an important tool in epidemiology, takes into account, many factors that bring about disease development and eradication, e.g transmission and recovery rate, mortality rate etc [1-3].

Vaccination is another well-known method in controlling disease spread [4,5]. But, it is impossible to vaccinate all susceptible individuals, especially in poor and third world countries where vaccines are not readily affordable. Clinically, vaccination brings about a temporary immunity to the disease, and once a vaccine wanes in a vaccinated individual, the vaccinated individual becomes susceptible to the disease again. The incidence rate is also an important factor in disease transmission, which is an effective contact between the susceptible and the infectious individual. Several authors have employed the use of non - linear incidence rate in describing qualitatively, the dynamics of disease transmission in human host population see [6-10].

Another important epidemic threshold is the basic reproduction number (R_0). It is established when cases of secondary infections arises when an infected individual is introduced into a host population of susceptible individual during the infected individual's lifetime [11,12]. R_0 helps in determining appropriate control measures to stop a disease spread, and it is obtained using the next generation matrix method [11]. Literatures of Proved useful in studying the local and global behavior of the linearizations of epidemic models around their equilibrium solutions [12-18].

Optimal control theory is an important tool in mathematics used in making decision involving complex epidemiological situations. Literature of worked on finding the best and effective optimum strategy to minimize disease spread [15,16,18,20]. In this paper, the cost of media coverage and treatment is used to minimize the number of infected individuals who become infected due to an epidemic breakout. The media coverage and treatment were used as control measures and the optimal intervention strategies for the disease control was established using the Pontryagin maximum principle (PMP). Also, the local and global stabilities of model system is investigated, and the geo- metric approach [21] is employed to analyze the global stability of the model at its endemic equilibrium solutions.

The rest of the paper is divided as follows; Section 2, involves the mathematical model formulation, positivity, and obtaining the two equilibria. In section 3, the basic reproduction number is obtained. Also, section 4, presents the local and global stability analysis for the disease-free and endemic equilibrium. While in section 5 the optimal control, existence of the control and the optimality of the system is studied and analyzed. Finally, section 6, deals with the numerical simulations and conclusion.

Mathematical Model Formulation

A first-order, deterministic model with non-linear incidence rate and constant recruitment A, is considered. The model is subdivided into compartmental state variables as, susceptible individuals (*S*), infected individuals (*I*) and vaccinated individuals (*V*). Also, several parameters incorporated into the model are, qA which represents the fraction of recruited individuals who are vaccinated into the susceptible population, *pS* is the fraction of vaccinated susceptible. Also, θ is the disease induced death rate for I compartment, represents the progression rate from *I* to *S* compartment, δ is the rate at which vaccine wanes thereby making vaccinated individuals γ to become susceptible to the disease again. μ is the natural death rate. Following the assumptions made and several state variables and parameters incorporated into the model formulation, the following differential equation governing the model is thus given as

$$\frac{dS}{dt} = (1 - q)A - \frac{\alpha S(t)I(t)}{1 + \beta S} - (p + \mu)S(t) + \gamma I(t) + \delta V(t),$$

$$\frac{dI}{dt} = \frac{\alpha S(t)I(t)}{1 + \beta S} - (\mu + \theta + \gamma) I(t), \tag{1}$$

$$\frac{dV}{dt} = qA + pS(t) - (\delta + \mu) V(t).$$

Subject to initial conditions $S(0) = S_0$, $I(0) = I_0$, $V(0) = V_0$.

In this model, the force of infection is taken as $C(I) = \frac{\alpha I(t)S(t)}{1+\beta S}$ where, β measures the infection forces of the disease and $1+\beta S$ measures the inhibition effect from the change in the behavior of the susceptible when their number increases.

Positivity, Boundedness and Equilibrium Solutions

The non - negative octant

$$\mathbb{R}^3_+ = \left[(S, I, V) \in \mathbb{R}^3 : \quad S \geq 0, \quad I \geq 0, \quad V \geq 0\right], \tag{2}$$

is positively invariant with respect to (1).

Theorem 1: All the solutions in (1) are uniformly bounded in the closed Set

$$\Delta = \left[(S, I, V) \in \mathbb{R}^3 : S + I \leq \frac{A}{\tau} \leq \frac{\rho A}{(\delta + \mu)\tau}\right]. \tag{3}$$

Proof. Let *(S (t), I (t), V (t))* be any solution with non negative initial conditions ($S_0 \geq 0$, $I_0 \geq 0$, $V_0 \geq 0$). From (1), we define a function

$$N(S, I) = S(t) + I(t). \tag{4}$$

Then

$$\frac{dN}{dt} \leq A - \tau N, \tag{5}$$

where $\tau = \min(\delta, \mu)$. Also

$$\frac{dN}{dt} + \tau N \leq A, \tag{6}$$

on applying the differential inequality theory, we obtain

$$N(S, I) = \frac{A}{\tau}(1 - \exp(-\tau t)) + N(S_0, I_0)\exp(-\tau t), \tag{7}$$

As $t \to \infty$, sup $N \leq \frac{A}{\tau}$. Furthermore, from the third equation in (1)

$$\frac{dV}{dt} = qA + \rho S - (\delta + \mu)V \leq \frac{\rho A}{\tau} - (\delta + \mu)V \cdot \tag{8}$$

Also, applying the standard comparison theorem, (8) yields

$$\lim \text{Sup}\, V \leq \frac{\rho A}{\tau(\delta + \mu)} \cdot \tag{9}$$

Therefore, as $\lim_{t\to\infty} V \leq \frac{\rho A}{\tau(\delta+\mu)}$ for all $t > 0$, since $S \geq 0$, $I \geq 0$, $V \geq 0$ then

$$\Delta = \left[(S, I, V) \in \mathbb{R}^3 : S + I \leq \frac{A}{\tau} \leq \frac{\rho A}{(\delta+\mu)\tau}\right]. \tag{10}$$

Hence, all the solutions of (1) starts and end in $\mathbb{R}^3_+$ and are restricted to Δ which is a bounded and a positively invariant region. Model system (1) will be studied in Δ since it is epidemiologically reasonable and mathematically well posed. This completes the proof.

In the interior of $\mathbb{R}^3_+$ there are two possible equilibrium solutions by equating the right hand side of (1) to 0 at when $I = V = 0$ and $I = V \neq 0$.

Theorem 2: In the interior of $\mathbb{R}^3_+$ there exists disease-free equilibrium points E^0 if $R_0 < 1$, and there exists endemic equilibrium points E^* if $R_0 > 1$.

Proof. Equating the right hand side of (1) to zero,

$$\begin{aligned} 0 &= (1-q)A - \tfrac{\alpha SI}{1+\beta S} - (\rho + \mu)S + \gamma I + \delta V\,, \\ 0 &= \tfrac{\alpha SI}{1+\beta S} - (\mu + \theta + \gamma)I\,, \\ 0 &= qA + \rho S - (\delta + \mu)V, \end{aligned} \tag{11}$$

such that when disease is absent from the system (11) i.e., $I = V = 0$, then

$$E^0 = \left(\frac{A}{\rho + \mu}\left(1 - \frac{C}{R_0(\mu + \theta + \gamma)}\right), 0, \frac{qA}{(\delta + \mu)}\left(\frac{1}{R_0}\right)\right). \tag{12}$$

Also, when disease is present in the system (11) i.e. $S = I = V \neq 0$ yields

$$\begin{aligned} E^* = (S^*, I^*, V^*) = \Bigg(\frac{1}{R_0}, &\frac{\gamma I + \delta V + (1-q)A - (\rho + \mu)(\mu + \theta + \gamma)}{\mu + \theta + \gamma}(R_0 - 1)\,, \\ &, \frac{CqA + \rho(\mu + \theta + \gamma)}{C(\delta + \mu)}(R_0 - 1)\Bigg). \end{aligned} \tag{13}$$

Reproduction Number (R_0)

The next generation operator method [7], is employed to obtain R_0. Say, $\dot{x} = f(x)$ where the components $f_i(x) = F_i(x) - V_i(x)$ for $i = 1,\ldots.. n$, then $F_i(x)$ is the rate of appearance of new infections in compartment i. While $V_j^-(x) - V_i^+(x)$, with $V_i^+(x)$ the rate of transfer of individuals into compartment i by any other means, and $V_i^-(x)$ the rate of transfer out of compartment i. Such that

$$F = \begin{pmatrix} 0 & 0 & \frac{CA}{\rho+\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} (\rho+\mu) & 0 & \delta \\ 0 & \mu+\theta+\gamma & 0 \\ \rho & 0 & (\delta+\mu) \end{pmatrix}. \tag{14}$$

Where

$$V^{-1} = \begin{pmatrix} \frac{\delta+\mu}{(\delta+\mu+\rho)\mu} & 0 & -\frac{\delta}{(\delta+\mu+\rho)\mu} \\ 0 & \frac{1}{\mu+\theta+\gamma} & 0 \\ -\frac{\rho}{(\delta+\mu+\rho)\mu} & 0 & \frac{\mu+\rho}{(\delta+\mu+\rho)\mu} \end{pmatrix}. \tag{15}$$

The R_0 as the spectral of a positive matrix is the largest eigenvalue given as

$$R_0\left(FV^{-1}\right) = \frac{CA}{(\rho+\mu)(\mu+\theta+\gamma)}. \tag{16}$$

Stability Analysis of the Model

Local Stability Analysis of Disease-Free Equilibrium

Theorem 3: The disease free equilibrium of (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 \geq 1$.

The Jacobian of (1) at disease free equilibrium solutions (11) is given as [14].

$$J = \begin{pmatrix} -(\rho+\mu) & M_1+\gamma & \delta \\ 0 & M_1-(\mu+\theta+\gamma) & 0 \\ \rho & 0 & -(\delta+\mu) \end{pmatrix}. \tag{17}$$

The characteristics polynomial of (17) is

$$(\mu+\lambda)(\mu+\theta+\gamma+\lambda-M_1)(\mu+\rho+\delta+\lambda), \tag{18}$$

where M1 is given as $\frac{CA}{p+\mu}$. Using the principle of the trace-determinant plane, the trace of (17) yields

$$-\delta-3\mu+M_1-\theta-\gamma-\rho>0, \tag{19}$$

and the characteristics equation for the remaining determinant is given by

$$\lambda^2+\lambda[q_1+q_2]+q_1q_2, \tag{20}$$

where

$$q_1 = M_1-(\mu+\theta+\gamma), \quad q_2 = 0. \tag{21}$$

Applying the Routh-Hurwitz principle, (17) has a strictly negative root if and only if $q_1 \leq 0$ and $q_2 \leq 0$ and $q_1 \geq q_2$. Then

$$\frac{CA}{(\mu+\theta+\gamma)(\mu+\rho)} > \frac{(\mu+\theta+\gamma)}{(\mu+\theta+\gamma)}, \tag{22}$$

this further shows that

$$R_0 - 1 > 0, \quad -R_0 > -1, \quad R_0 < 1. \tag{23}$$

Hence, the disease-free equilibrium is locally aysmptotically stable.

Global Stability of Disease-Free Equilibrium

Theorem 4: The disease-free equilibrium of system (1) is globally asymptotically stable if $R_0 < 1$ [15,16].

Proof. Given that $R_0 < 1$, there exists only the disease free equilibrium E^0.

Consider a Lyapunov function candidate $V(S, I, V): \mathbb{R}^3 \to \mathbb{R}^+$ defined as

$$V(S, I, V) = \eta I, \quad \eta \geq 0. \tag{24}$$

Substituting the second equation in (1), yields

$$\dot{V} = \eta[(CS - (\mu + \theta + \gamma))I], \tag{25}$$

since $S^0 = \frac{A}{\rho + \mu}$, $C = \frac{\alpha}{1 + \beta S}$, let $\eta = \frac{1}{(\mu + \theta + \gamma)}$. At when $I = 0$,

$$\dot{V} = \eta \left(\frac{CA - (\rho + \mu)(\mu + \theta + \gamma)}{(\rho + \mu)} \right) I, \tag{26}$$

$$\dot{V} = [R_0 - 1] I \leq 0. \tag{27}$$

Since all the parameters and variables involved in the model system (1) are all positive constants, then $V \leq 0$ for $R_0 < 1$ and $V = 0$ if and only if $I = 0$. Hence, the disease-free steady-state is globally asymptotically stable.

Local Stability of Endemic Equilibrium

Theorem 5: The endemic equilibrium point is locally asymptotically stable if and only if $R_0 > 1$ [14].

The Jacobian matrix of (1) at the endemic equilibrium points (12) is yields

$$J = \begin{pmatrix} -CI^* - (\rho + \mu) & CS^* + \gamma & \delta \\ CI^* & CS^* - (\theta + \mu + \gamma) & 0 \\ \rho & 0 & -(\delta + \mu) \end{pmatrix}, \tag{28}$$

The characteristics polynomial of (28) yields

$$\lambda^2 + \lambda^2 K_1 + \lambda K_2 + K_3. \tag{29}$$

Where

$$K_1 = 3\mu + \delta + \theta + \gamma + \rho + CI^* - CS^*, \tag{30}$$

$$K_2 = \delta C^* + CS^*(2\mu + \rho) + CI^*(2\gamma + \theta + \delta + 2\mu) - 2\mu(\theta + \delta + \rho) - \theta(\delta + \rho) - 3\mu^2, \tag{31}$$

$$K_3 = \delta C^* + CS^*(2\mu + \rho) - CI^*\left(2\gamma\delta + 2\gamma\mu + \theta\delta + \theta\mu + \delta\mu + \mu^2\right) + 2\gamma\left(\delta\mu + \mu^2 + \mu\rho + \delta\gamma\right) + \theta\left(\delta + \mu^2 + \gamma\rho\right) + \mu^2(\mu + \rho). \tag{32}$$

It is observed from (29 - 31) that, $K_1 > 0$, $K_2 > 0$, K_3 $(1\text{-}R_0) > 0$. Hence, $K_1K_2 - K_3 > 0$. by the Routh-Hurwitz criteria, $R_0 > 1$ implies that the endemic equilibrium is locally asymptotically stable [18].

Global Stability of Endemic Equilibrium

In this section, the Li and Muldoney, approach is employed [21].

Theorem 6: Let $Z \in R^n$ n be an open set and $f: x \to f(x) \in R^n$ be C^1 function for x in an open se $\Delta \in R^n$. Consider the differential equation.

$$x' = f(x), \tag{33}$$

Where $x(t, x_0)$ denotes the solution of (33) satisfying $x(0, x_0) = x_0$.

The following two assumptions were made.

i There exists a compact absorbing set $K \in \Delta$.

ii The solution (32) has a unique equilibrium $\overline{x}$ in Ω .

Let $Q: x \to Q(x)$ be an

$$\binom{n}{2} \times \binom{n}{2} l, \tag{34}$$

matrix valued function. That is, C^1 and Q^{-1} (x) exists for $x \in \Delta$. Let μ be a Lozinskii measure on $R^{d\times d}$, where $d = \binom{n}{2}$. Define a quantity q_2 as

$$\overline{q}_2 = \lim_{t\to\infty} Sup_{x_0\in\Delta} \frac{1}{t} \int_0^1 \mu(M(x(s, x_0)))\, ds, \tag{35}$$

where, $M = Q_f Q^{-1} + QJ^{[2]}Q^{-1}$. The matrix Q_f is obtained by replacing each entry q_{ij} of Q by its derivative in the direction of f_i, (q_{ij}) and $J^{[2]}$ is the Second additive compound matrix of the Jacobian Matrix J of (33).

Proof. By the theorem above, if $R_0 > 1$, then, E^* is the unique endemic equilibrium in the interior of Δ. The persistence of (1) coupled with its bounded solutions shows that the compact absorbing set Δ exists. The Jacobian matrix J of (1) is obtain as

$$J = \begin{pmatrix} CI - (\rho + \mu) & CS + \gamma & \delta \\ CI & CS - (\theta + \mu + \gamma) & 0 \\ \rho & 0 & -(\delta + \mu) \end{pmatrix}. \tag{36}$$

The second additive compound matrix of (36) is

$$J^{[2]} = \begin{pmatrix} CI + CS - (\rho + 2\mu + \theta + \gamma) & CS - (\mu + \theta + \gamma) & -\delta \\ 0 & CS - 2\mu - (\rho + \delta) & CS - \gamma \\ -\rho & CI & CS - 2\mu - (\mu + \theta + \gamma) \end{pmatrix}. \tag{37}$$

Let the function

$$P(x) = P(S, I, V) = \text{diag}\left(1, \frac{I}{V}, \frac{I}{V}\right), \tag{38}$$

where

$$P^{-1}(x) = \left(1, \frac{I}{V}, \frac{I}{V}\right), \tag{39}$$

and

$$P_f = \left(0, \frac{\dot{I}}{V} - \frac{I\dot{V}}{V^2}, \frac{\dot{I}}{V} - \frac{I\dot{V}}{V^2}\right), \tag{40}$$

so that

$$P_f P^{-1} = \left(0, \frac{\dot{I}}{I} - \frac{\dot{V}}{V}, \frac{\dot{I}}{I} - \frac{\dot{V}}{V}\right), \tag{41}$$

Also,

$$q = P_f P^{-1} + PJ^{[2]} = \begin{pmatrix} q_{11} & q_{12} \\ q_{21} & q_{22} \end{pmatrix}, \tag{42}$$

such that (41) written as a block form yields

$$q_{11} = CI + CS - (\rho + 2\mu + \theta + \gamma), \quad q_{12} = CS - (\mu + \theta + \gamma)\frac{1}{V} - \delta\frac{1}{V},$$

$$q_{21} = \left[0, \rho\frac{I}{V}\right]^T, q_{22} = \begin{pmatrix} \frac{i}{I} - \frac{\dot{V}}{V} - CI - 2\mu - (\rho + \delta) & CS + \gamma \\ CI & \frac{i}{I} - \frac{\dot{V}}{V} - CS - 2\mu - (\theta + \gamma + \delta) \end{pmatrix}. \tag{43}$$

Let (u, v, w) be the vectors in $\mathbb{R}^3$, we select a norm in $\mathbb{R}^3$ as |(u, v, w)| = $\max\left[|u|, |u+w|\right]$ and let μ be the Lozinskii measure with respect to this norm, such that

$$\mu(q) \leq \sup(h_1, h_2), \tag{44}$$

Where

$$h_1 = \mu_1(q_{11}) + |q_{12}| \tag{45}$$

and

$$h_2 = (q_{21}) + \mu_1 |q_{22}|, \tag{46}$$

are the matrix norms with respect to l_1 vector norm and is the lozinskii measure with respect to l_1 norm. Also,

$$\mu_1(q_{11}) = CI + CS - (\rho + 2\mu + \theta + \gamma), \quad q_{21} = \rho\frac{I}{V}, \tag{47}$$

$$\mu_1(q_{12}) = \max\left[CS - (\mu + \theta + \gamma), \delta\frac{I}{V}\right] = (CS - (\mu + \theta + \gamma), -\delta)\frac{I}{V}, \tag{48}$$

$$\mu_1(q_{22}) = \max\left[\frac{\dot{I}}{I} - \frac{\dot{V}}{V} - CI - 2\mu - (\rho + \delta), -CS - 2\mu - (\theta + \gamma + \delta), CI\right], \tag{49}$$

$$= -2\mu - \delta + \frac{\dot{I}}{I} - \frac{\dot{V}}{V} + \max[-\rho, -\gamma, -\theta].$$

Therefore, $h_1 = \mu(q_{11}) + |q_{12}|$, where

$$\begin{aligned} h_1 &= CI + CS - (\rho + 2\mu + \theta + \gamma) + CS - (\mu + \theta + \gamma), -\delta), \\ &\leq \frac{\dot{I}}{I} - (\mu + \theta + \gamma) + CS = \frac{\dot{I}}{I} - \mu, \end{aligned} \tag{50}$$

and

$$h_2 = |q_{21}| + \mu_1 (q_{22}),$$
$$h_2 = -2\mu\delta + \frac{i}{I} - \frac{\dot{V}}{V} + \max[-\rho, -\gamma, -\theta] \leq \frac{i}{I} - 2\mu - \delta, \quad (51)$$

so that

$$\mu q \leq \sup(h_1, h_2) \leq \frac{\dot{I}}{I} - \mu. \quad (52)$$

Then

$$d_1 = \frac{1}{t}\int_0^t \mu q ds \leq \frac{1}{t}\int_0^t \left(\frac{\dot{I}}{I} - \mu\right) ds = \frac{1}{t}\ln\frac{I(t)}{I(0)} - \mu, \quad (53)$$

implies that $d_1 = q \leq \frac{-\mu}{2} < 0$. Thus, the endemic equilibrium E^* of (1) is globally asymptotically stable.

Optimal Control

The controls imposed on (1) are the use of treatment and media coverage, such that $0 \leq u_1 \leq 1$ and $0 \leq u_2 \leq 1$. The controlled model is given by

$$\begin{aligned}
\frac{dS}{dt} &= (1-q)A - \frac{\alpha S(t)I(t)}{1+\beta S} - (\rho+\mu)S(t) + \gamma I(t) + \delta V(t) - \mu_2 S(t),\\
\frac{dI}{dt} &= \frac{\alpha S(t)I(t)}{1+\beta S} - (\mu+\theta+\gamma)I(t) - \mu_1 I(t),\\
\frac{dV}{dt} &= qA + \rho S(t) - (\delta+\mu)V(t) + \mu_1 I(t) + \mu_2 S(t),
\end{aligned} \quad (54)$$

subject to initial conditions $S(0) \geq 0, I(0) \geq 0, V(0) \geq 0$.

The objective functional is defined as

$$J(u_1, u_2) = \int_0^T \left(L_1 + L_2\frac{u_1^2}{2} + L_3\frac{u_2^3}{3}\right) dt. \quad (55)$$

While, the control set U is Lebesgue measurable and defined as

$$[(u_1(t), u_2(t)|0 \leq u_1 \leq 1, \quad u_2 \leq 1, \quad t \in [0, T])]. \quad (56)$$

Also, a quadratic, non-linear cost control is employed to analyze the behavior of the cost implementation of the treatments and media coverage. The goal is to minimize the total number of infectious individual and at the same time minimize the cost of treatment and media coverage in the host population. L_1 denotes the total number of infectious individual taken as a measure of death in the event of epidemic breakout, $L_2 \frac{u_1^2}{2}$ denotes the cost of treatments,while $L_3 \frac{u_2^3}{3}$ denotes the cost of media coverage.

Moreover, L_1, L_2 and L_3 are the relative weights attached to the minimization cost of the total number of infectious individual, treatment cost and media coverage cost respectively.

Existence and Uniqueness of the Control

Theorem 7: If the objective functional

$$J(u_1, u_2) = \min[J(u_1, u_2)] = \int_0^1 \left(L_1 I + L_2\tfrac{u_1^2}{2} + L_3\tfrac{u_2^3}{3}\right) dt,$$

where $u = [u_1; u_2 : 0 \leq u_1(t) \leq 1, 0 \leq u_2(t) \leq 1, t \in [t_0; T] \in < \mathbb{R}^+$, subject to constraints of (54) and (55) with

S(0) = S_0, I(0) = I_0 and R(0) = R_0, there exists optimal control $u_1^* = (u_1^*, u_2^*)]$, such that $\min_{u_1 u_2 \in u} J(u_1, u_2) = J(u_1^*, u_2^*)$

Proof. According to fleming and rischel [22], the control and state variables are non-negative, and the two control variable u_1, u_2 ε U is closed and convex. Also, the integrand in the objective functional defined as $\left(L_2\frac{u_1^2}{2} + L_3\frac{u_2^3}{3}\right)$ is convex on *U*.

There exists constants b_1,$b_2 > 0$ and $\beta > 1$ such that $\left(L_2\frac{u_1^2}{2} + L_3\frac{u_2^3}{3}\right)$ is convex and satisfy $J(u_1, u_2) \geq b_1\left(\left|\frac{u_1^2}{2}\right|^2 + \left|\frac{u_2^2}{2}\right|^2\right)^{\frac{\beta}{2}} - b_2$.

This implies that

$$u_1^* = \left\{ \begin{array}{ll} 0 \text{ if} & z_1^* \leq 0 \\ z_1^* \text{ if} & 0 \leq z_1^* \leq 1 \\ 1 \text{ if} & z_1^* \geq 0 \end{array} \right\}, \tag{57}$$

where

$$z_1^* = \frac{(\lambda_I - \lambda_V) I}{L_2}, \tag{58}$$

$$u_2^* \left\{ \begin{array}{ll} 0 \; if & z_2^* \leq 0 \\ z_2^* \; if & 0 \leq z_2^* \leq 1, \\ 1 \; if & z_2^* \geq 0 \end{array} \right\}, \tag{59}$$

where

$$z_2^* = \frac{(\lambda_S - \lambda_V) S}{L_3}. \tag{60}$$

Hence, by the boundedness of the state and adjoint system, the optimal control exists and is unique for small *T*.

Theorem 8. Given that $U^*(t) = (u_1^*, u_2^*)$ and $X^*(t) = (S^*(t), I^*(t), V^*(t))$ of (54), there exists adjoint variables $\lambda_S(t)$, $\lambda_I(t)$ and $\lambda_V(t)$. The derived hamiltonian is given by

Proof. $H(S, I, V) = \left(L_1 I + L_2\frac{u_1^2}{2} + L_3\frac{u_2^3}{3}\right) + \lambda_S((1-q)A - CSI - (\rho + \mu)S + \gamma I + \delta V - \mu_2 S(t)) + \lambda_I(CSI - (\mu + \theta + \gamma)I - \mu_1(t)) + \lambda_V(qA + \rho S - (\delta + \mu)V + \mu_1 I(t)) + \mu_2 S(t))$,

so that

$$\lambda_S = \lambda_S\left(CI - (\rho + \mu) - \mu_2\right) - \lambda_I(CI) - \lambda_V\left(\rho + \mu_2\right), \tag{61}$$

$$\lambda_I = L_1 + \lambda_S CS + \lambda_S\gamma + \lambda_I(CS - \mu + \theta + \gamma) + \lambda_V\mu, \tag{62}$$

$$\lambda_V = \lambda_S\delta + (\delta + \mu)\lambda_V, \tag{63}$$

with final condition $\lambda_S(T) = \lambda_I(T) = \lambda_V(T) = 0$.

The optimal control u_1^* and u_2^*, are respectively given as

$$u_1^* = \min\left[\max\left(0, \frac{\lambda_I - \lambda_V I}{L_2}, 1\right)\right], \tag{64}$$

$$u_2^* = \min\left[\max\left(0, \frac{\lambda_S - \lambda_V S}{L_3}, 1\right)\right]. \tag{65}$$

Optimality System

The optimality system of the state equations are

$$\begin{aligned} \frac{dS}{dt} &= (1-q)A - \frac{\alpha S(t)I(t)}{1+\beta S} - (\rho+\mu)S(t) + \gamma I(t) + \delta V(t) - \mu_2 S(t) \\ \frac{dI}{dt} &= \frac{\alpha S(t)I(t)}{1+\beta S} - (\mu+\theta+\gamma)I(t) - \mu_1 I(t) \\ \frac{dV}{dt} &= qA + \rho S(t) - (\delta+\mu)V(t) + \mu_1 I(t) + \mu_2 S(t) \end{aligned}, \tag{66}$$

and

$$S(0) \geq 0, \quad I(0) \geq 0, \quad V(0) \geq 0\,. \tag{67}$$

While the adjoint equations are

$$\dot{\lambda}_S = -\frac{\partial H}{\partial S} = -\left[\lambda_S\left(-CI - (\rho+\mu) - \mu_2\right) + \lambda_2(CI) + \lambda_V\left(\rho+\mu_2\right)\right], \tag{68}$$

$$\dot{\lambda}_I = -\frac{\partial H}{\partial I} = -\left[L_1 + \lambda_S(CS+\gamma) + \lambda_I\left(CS - (\mu+\theta+\gamma) + \lambda_V\left(\mu_1\right)\right)\right], \tag{69}$$

$$\dot{\lambda}_V = -\frac{\partial H}{\partial V} = -\left[\lambda_S(\delta) + (-(\delta+\mu))\lambda_V\right], \tag{70}$$

with the transversality conditions as,

$$\lambda_S(T) = \lambda_I(T) = \lambda_R(T) = 0. \tag{71}$$

Numerical Simulations

Numerical simulations and graphical illustrations is performed in order to validate the analytical results of this study. Different initial starts and parameter values have been used to obtain the graphical view of the results. The optimal control numerical algorithm is done with the aid of MATLAB software using the forward-backward sweep technique. Also the optimality of the system solved using the fourth-order Runge-Kutta.

Variable	Descriptions	Values	Source
S(0)	Susceptible Individuals	50	Assumed
I(0)	Infected Individuals	20	Assumed
V(0)	Vaccinated Individuals	10	Assumed

Table 1: State Variables in Model (1) and their Meanings

Parameters	Descriptions	Values	Source
qA	Vaccinated Recruited Susceptibles	0.45 persons/day^{-1}	Estimated
α	Transmission coecient	1.112 persons/day^{-1}	Estimated
β	Transmission rate	0.213 persons/day^{-1}	Estimated
ρ	Vaccinated Susceptibles	0.07 persons/day^{-1}	Estimated
μ	Natural death rate	0.009 persons/day^{-1}	Estimated

γ	Progression rate	0.11 persons/day^{-1}	Estimated
δ	Rate at which vaccine wanes	0.03 persons/day^{-1}	Estimated
c	Incidence rate	1.431 persons/day^{-1}	Estimated
θ	Disease induced death rate	1.22 persons/day^{-1}	Estimated

Table 2: Parameters in Model (1) and their Meanings

Figure 1: Describes the behavior of the solution curve of state variable S and I for time interval [0; 0:12]. The Runge Kutta 4th order iterative method is used to solve the state variables forward in time. See [19]

Figure 1: Graph of Optimality system S and I against time(t)

Figure 2: Describes the behavior of the solution curve of state variable V for time interval [0; 3]. The Runge Kutta 4th order iterative method is used to solve the state variable V forward in time. See [19]

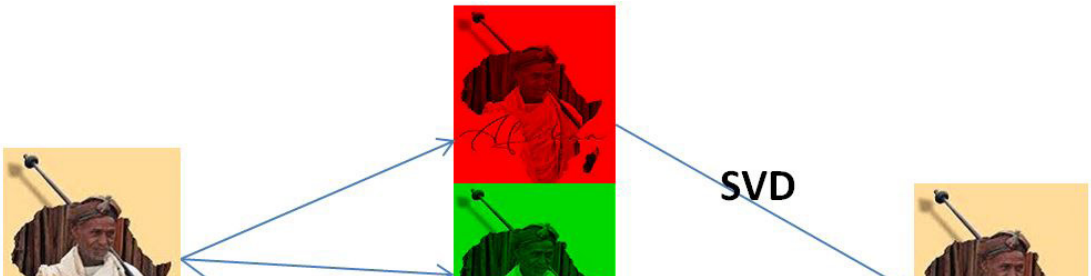

Figure 2: Graph of optimality system of V against time (t)

Figure 3: Presents the impact of the of the two controls on the susceptible individuals. The media coverage control is not effective enough because it doesn't tend to zero unlike the treatment control (u_2), with high capacity to minimize the number of infectives and bring about the absolute eradication of the disease in human host population.

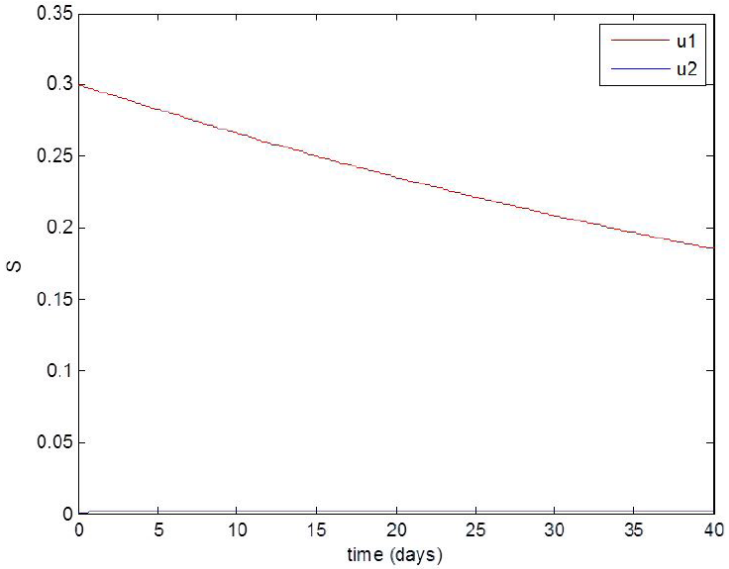

Figure 3: Graph of S with two controls u1 and u2 against time (t)

Figure 4: Shows the behavior of the Vaccinated class. The impact of media coverage information control is more felt by making more individuals to make themselves readily available for vaccination through information. This brings about the eradication of disease in the human host population.

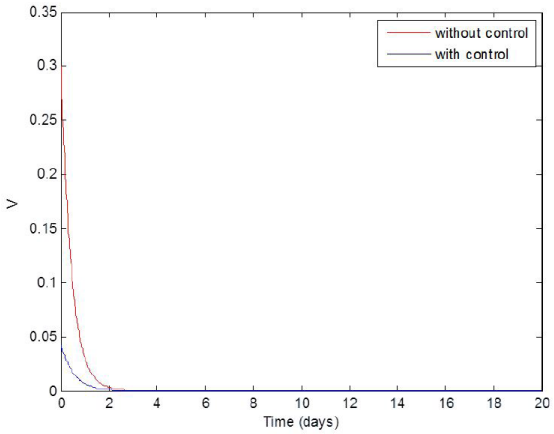

Figure 4: Graph of V with and without control against time (*t*)

Figure 5: Describes the behavior of the adjoint equations λ_S; λ_I ; λ_V . The adjoint equations were solved backward in time using the Runge-Kutta 4th order iterative technique. The values of the adjoint tend to zero towards the final time. This implies that the rate of change of Hamiltonian H increases with respect to the state variables S, I, V.

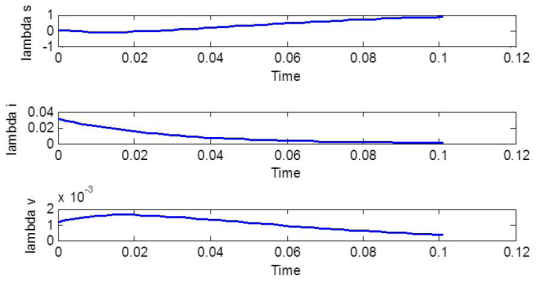

Figure 4: Graph of λ_s;λ_I ; λ_V against time (*t*)

Figure 6: Presents the impact of control and absence of control on the infected class. When the treatment control is applied, there is a decline, tending to zero, showing that the number of infected individuals is reduced to the barest minimum, thereby leading to the total eradication of the disease in the human host population.

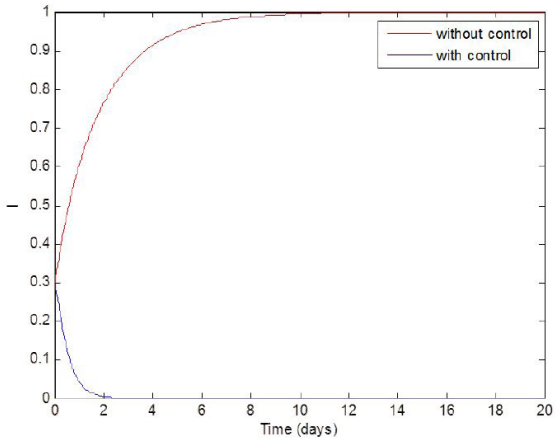

Figure 6: Graph of I with and without control against time

Conclusion

In this paper, a deterministic system of differential equation of SIS -V S is presented in order to gain insight into the epidemic transmission in human host population and bring about effective optimum strategies for the control of the disease spread. The conditions for the optimal control of the disease with media coverage and treatment measures is derived and analyzed, it is observed that the media coverage and treatment have a great effect in combating any infectious diseases. Also, the local and global analysis is carried out on the model to study the existence and stability of the disease free and endemic equilibrium. When R0 < 1, the disease free equilibrium exists and it is both locally and globally stable. Also, if R0 > 1, the endemic equilibrium is locally and globally stable. The model considered in this paper can be extended by incorporating time and space thereby changing it from ordinary differential equation to partial differential equations. Also, the cost effectiveness of the controls is another area that can be effectively analyzed and studied.

References

1. Anderson RM, May RM (1992) Infectious disease of humans: Dynamics and control, Oxford univ press 15: 126.

2. Berretta E, Capasso V (1986) On the general structure of epidemic systems: Global asymptotic stability. Comp Math Appl part A 12 677-94.

3. Hethcote HW (1994) A thousand and one epidemic model. front.theor.biol 100: 504-15.

4. Chan L, Fan M, Feng ZL (2011) Dynamics of a multigroup epidemiological model with group - targeted vaccination strategies. J Theor Biol 291: 56-64.

5. Teitelbaum MA, Edmunds M (1999) Immunization and Vaccine Preventable Illness, USA 1992 - 1997.Statist Bull Metrop Insur Co 80: 13-20

6. Kaddar A (2009) On the dynamics of delayed SIR epidemic model with a modified saturated incidence rate. J Differ Eqn 133: 1-7.

7. De-leheenher P, Smith HL(2003) Virus dynamics: A global analysis. SIAM J Appl Math 63: 1313 -27.

8. Xiao D, Ruan S (2003) Dynamical behavior of an epidemic model with non -linear incidence rate. J differ eqtn 188: 135-63.

9. Alexander ME, Moghadas SM (2004) Periodicity in an epidemic model with generalized non linear incidence. Math Biosci159: 75-96.

10. Hou J, Zeng Z (2009) Continuous and impulsive vaccination of SEIR epidemic models with saturation incidence rate. Math Comput Simul 79: 3038-54.

11. Diekmann O, Heesterbeek JAP, Roberts MG (2010) Construction of next generation matrices for compartmental models in epidemics, J royal soc of biol inter 7: 875-85.

12. Van den Driessche P, Watmough J (2002) Reproduction number and sub threshold endemic equilibria for compartmental models of disease transmission. Math Biosci 180: 29-48.

13. Cull P (1986) Local and global stability for population models Biological cybernetics. 54: 141-9.

14. Esteva-Peralta L, Velasco-Hernandez JX (2002) M-Matrices and Local Stability in Epidemic Models. Math Comput Modell 36: 491-501.

15. Korobeinikov A (2004) Lyapunov functions and global stability for SEIR and SEIS epidemiological models. Math Med Biol 21: 75-83.

16. Korobeinikov A, Wake GC (2002) Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. Appl Math Letters 15: 955-60

17. la - Salle J, Lifschetz S (1962) Stability by Lyapunov direct method with applications, Acad press 59.

18. Shahu B.K. Stability Analysis of Non Linear Systems Using Dynamic Routh-Hurwitz Criteria.

19. Thieme RH (1993) Persistence under relaxed point - dissipativity with application to an endemic model, SIAM J Math Anal 24: 407-35.

20. Lehnhart SM, Workman JT (2007) Optimal control applied to biological Models. CRC press 274.

21. Li MY, Muldoney JS (1996) Geometric approach to global stability problems Siam J Math Anal 27: 1070-83.

22. Fleming, Wendell H, Rishel, Raymond W (1995) Deterministic and Stochastic Optimal Control. Springer - Verlag, New york.

23. Fister K, Donelly J (2005) Immunotherapy: An optimal control theory approach. Math Biosci Eng 499 -510.

24. Gaff H, Schaefer E (2010) Optimal control applied to vaccination and treatment strategies for various epidemiological models. J R Soc boil interface 7: 875-85.

25. Josh HR (2002) Optimal control models in PDE and ODE systems. Thesis of Doctorate, university of tennessee, USA.

26. Pontryagin LS (1962) The mathematical theory of optimal processes. John Wiley and sons UK.