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Research Article

Mathematical Modeling and Stability Analysis of a *SIRV* Epidemic Model with Non-linear Force of Infection and Treatment

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Abstract. This paper considers the *Susceptible-Infected-Vaccinated-Recovered* (*SIRV*) deterministic model with a non linear force of infection and treatment, where individual humans that are vaccinated losses their vaccination after some time and become vulnerable to infections. The basic reproduction number R_0 obtained from the model system is an epidemic threshold that determines if a disease will continue to ravage the human population or not. The model state equations considered in this paper possess two steady-state solutions such that if $R_0 < 1$, the infection-absent steady-state solutions are locally and globally asymptotically stable. Also, if $R_0 > 1$, a unique infection-persistent steady-state solutions are established, which is also locally and globally asymptotically stable. Thus, it leads to the persistence of infections in the human host population. Finally, numerical simulations were carried out to validate our theoretical results.

Keywords. Vaccination; Local stability; Reproduction number; Steady states; Global stability

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1. Introduction

In the study of epidemics, vaccination is an indispensable control strategy to prevent and eliminate diseases in a human host population [3], [16]. Efficacious vaccines protects susceptible human individual if it is applied on time before exposure to the disease. Many apparent and

re-appearing diseases have been combated with appropriate vaccinations e.g. rubella, measles, cholera, malaria, Hepatitis A and B, etc. Mathematical models have been formulated in several articles to describe how diseases are transmitted within compartments of sub-populations of human, where individuals are grouped into compartments according to the characteristics and purpose of the disease being modeled. Some of the authors who have employed mathematical techniques to describe the transmission dynamics of a disease includes ([1], [4], [5], [6], [7], [8], [12], [14], [15], [17], [18]).

However, in the model formulation of a classic SIR model, vaccination class is included so that after the recovery of a sick individual from a disease, appropriate vaccines can be administered to the recovered and the susceptible individuals in the host population. In epidemiology, incidence force of infection is simply defined as the rate at which individuals are being infected per unit time when an infected individual come in contact with a susceptible individual during his or her period of infections. The law of mass action incidence (βSI), where β as a transmission parameter is earlier used, but could not take into account of disease outbreak in larger populations. This has led to the formulations of nonlinear forces of infection by some authors [10]. Moreover, asymptotic stabilities independent of initial conditions are shifted from the infection-absent to the infection-present steady-state. To investigate the global properties of a epidemic model system is non-trivial because there are no known mathematical methods for constructing Lyapunov functions for epidemic models. Systematic method of direct Lyapunov or nonlinear Lyapunov function of the Goh-Volterra type are good approaches to obtaining it ([2], [19]).

Having gone through [9], [11], [13], in our work, we formulated a SIRV model with variable size population. Instead of the mass action incidence, by extension, we incorporated a non linear incidence force of infection of the form $\frac{\alpha SI}{1+\beta S}$, which account for the contact between susceptible and infected individual leading to overcrowding because of high level of saturation of the disease. Also, nonlinear treatment rate (rI) and other parameters are included in the model build up. The rest of the paper is organized as follows. Section 2 presents the model formulation, existence and uniqueness of the model solutions, positivity of the system, existence of the state equations steady-solutions and the basic reproduction number (R_0). In Section 3, the local and global asymptotic stability of the infection absent steady-state is investigated. Section 4 involves the analysis of the local and global asymptotic stability of the infection persistent steady-state. Finally, Section 5 presents the numerical simulations and conclusion.

2. Model Formulation and Analysis

In this section, we consider a epidemic transmission model based on a deterministic, non linear, first order system of ordinary differential equations. The total host population $N(t)$ is subdivided into sub-populations of state variables of individuals who are susceptible individuals $S(t)$, infected individuals $I(t)$, recovered individuals $R(t)$, and vaccinated individuals $V(t)$, so that $N(t) = S(t) + I(t) + R(t) + V(t)$. Humans are recruited into the population at a rate A. There is an effective infectious contact between the susceptible and infected individual at a rate $\frac{\alpha}{1+\beta S}$ represented by λ. ρ is rate at which a certain fraction of susceptible individuals receives vaccination, μ is the natural death rate applicable to all compartments. δ_1 and δ_2 are the rates at which the recovered and vaccinated compartments losses their immunities to treatment and vaccination respectively. Also, α is the death rate induced by infections of infected individuals,

while γ is the natural recovery rates due to other factors and rI is the treatment rate of the infected class. The following assumptions is made in the model build up that,

i. Birth and death rate is certain.
ii. Susceptible Individuals are infected if they come in contact with an infected individual except those that are vaccinated.
iii. Vaccine losses its potency leading to waning in individuals after some time.
iv. An infected individual recovers after treatment.
v. There is no permanent recovery.
vi. There is homogenous mixture in the population.

Following the assumptions made, coupled with the state variables and parameters incorporated into the model, we now have the model system equations as

$$\begin{aligned}
\dot{S} &= A-\mu S-\lambda SI-\rho S+\delta_1 R+\delta_2 V,\\
\dot{I} &= \lambda SI-(\mu+\alpha+\gamma)I-rI,\\
\dot{R} &= \gamma I-\mu R-\delta_1 R,\\
\dot{V} &= \rho S-\mu V-\delta_2 V.
\end{aligned} \tag{1}$$

Subject to initial conditions $S(0)=S_0$, $I(0)=I_0$, $R(0)=R_{00}$, $V(0)=V_0$.

2.1 Existence and Uniqueness of Solutions of (1)

Theorem 2.1 ([5]). *Let* Ω *denote a region*

$$|t-t_0|\le y,\quad \|x-x_0\|\le z,\quad x=(x_1,x_2,\ldots,x_n),\quad x_0=(x_{10},x_{20},\ldots,x_{n0}). \tag{2}$$

Also, suppose the Lipschitzian condition $\|f(t,x_1)-f(t,x_2)\|\le c\|x_1-x_2\|$ *is satisfied by* $f(t,x)$*, whenever* (t,x_1) *and* (t,x_2) *is in* Ω*, where* c *is positive. A unique continuous vector solution* $x(t)$ *of the system in the interval* $t-t_0\le\delta$ *exists, such that* $\delta>0$.

Proof. Let Ω denote the region $0\le\alpha\le R$, we want to show that the partial derivatives of (1) are continuous and bounded in Ω. Let

$$\begin{aligned}
H_1 &= A-\mu S-\lambda SI-\rho S+\delta_1 R+\delta_2 V,\\
H_2 &= \lambda SI-(\mu+\alpha+\gamma)I-rI,\\
H_3 &= \gamma I-\mu R-\delta_1 R,\\
H_4 &= \rho S-\mu V-\delta_2 V.
\end{aligned} \tag{3}$$

Then the partial derivatives of (3) are given below as

$$\left|\frac{\partial H_1}{\partial S}\right|=|-(\mu+\lambda I+\rho)|<\infty,\quad \left|\frac{\partial H_1}{\partial I}\right|=|-\lambda S|<\infty,\quad \left|\frac{\partial H_1}{\partial R}\right|=|\delta_1|<\infty,\quad \left|\frac{\partial H_1}{\partial V}\right|=|\delta_2|<\infty, \tag{4}$$

$$\left|\frac{\partial H_2}{\partial S}\right|=|\lambda I|<\infty,\quad \left|\frac{\partial H_2}{\partial I}\right|=|\lambda S-(\mu+\alpha+\gamma)-r|<\infty,\quad \left|\frac{\partial H_2}{\partial R}\right|=|0|<\infty,\quad \left|\frac{\partial H_2}{\partial V}\right|=|0|<\infty, \tag{5}$$

$$\left|\frac{\partial H_3}{\partial S}\right|=|0|<\infty,\quad \left|\frac{\partial H_3}{\partial I}\right|=|\gamma|<\infty,\quad \left|\frac{\partial H_3}{\partial R}\right|=|-(\mu+\delta_1)|<\infty,\quad \left|\frac{\partial H_3}{\partial V}\right|=|0|<\infty, \tag{6}$$

$$\left|\frac{\partial H_4}{\partial S}\right|=|\rho|<\infty,\quad \left|\frac{\partial H_4}{\partial I}\right|=|0|<\infty,\quad \left|\frac{\partial H_4}{\partial R}\right|=|0|<\infty,\quad \left|\frac{\partial H_4}{\partial V}\right|=|-(\mu+\delta_2)|<\infty. \tag{7}$$

From (4), (5), (6), (7), it is clearly shown that the partial derivatives of (1) exists, are finite and bounded. Hence (1) has a unique solution. □

2.2 Positivity of (1)

Theorem 2.2. *If* $S(0)$, $I(0)$, $R(0)$, $V(0)$ *are nonnegative, then* $S(t)$, $I(t)$, $R(t)$, $V(t)$ *are also nonnegative for all time* $t > 0$.

Proof. The sum of all the system equations in (1) yield

$$\dot{N} = A - (S + I + R + V)\mu - \alpha I - rI, \tag{8}$$

such that in the absence of infections we have

$$\dot{N} = A - N\mu, \tag{9}$$

so that upon integration of (9) at both sides yields,

$$N = \frac{A}{\mu} + Ce^{-\mu t}, \tag{10}$$

where C is a constant. Then

$$N(t) = \lim_{t\to\infty}\left(\frac{A}{\mu} + \frac{C}{e^{\mu t}}\right) = \frac{A}{\mu}, \tag{11}$$

$$\limsup_{t\to\infty} N(t) \le \frac{A}{\mu}, \tag{12}$$

such that $N(0) \le \frac{A}{\mu}$. Then the feasible region is given by

$$\Omega = \left[(S,I,V,R) \in \Re_+^4; N(t) \le \frac{A}{\mu}, S + I + R + V \le \frac{A}{\mu}\right]. \tag{13}$$

This show that $\frac{A}{\mu}$ is the upper bound while 0 is the lower bound of (1). Therefore, Ω is positively invariant and the model system (1) is well posed mathematically and realistic in an epidemic sense. □

2.3 Existence of Steady State Solutions

The existence of the steady state-solutions is carried out in order to investigate the long term behavior of (1) which largely depends on R_0 and its steady-state solutions. The model considered in this paper posses two steady-state solutions. In order to obtain the steady-state, model system (1) is made static i.e., obtain the time-independent solutions of the model. The steady-state solutions in the absence of infections i.e., $I = 0$ is given by

$$E^0 = (S,I,V,R) = \left(\left(\frac{A}{\rho+\mu}\right)\left(1 - \frac{\lambda}{R_0(\mu+\alpha+\gamma)-r}\right), 0, 0, \left(\frac{\rho}{\mu+\delta_2}\right)\left(\frac{1}{R_0}\right)\right). \tag{14}$$

Also, the steady-state solutions when infection is persistent i.e., $I \neq 0$ is given by,

$$\begin{aligned} E^* = (S^*,I^*,R^*,V^*) = \Bigg(&\left(\frac{A+\delta_1 R^*+\delta_2 V^*}{\mu-\lambda I^*+\rho}\right)\left(\frac{1}{R_0}\right), \frac{\lambda(A+\delta_1 R^*+\delta_2 V^*)}{(\mu-\lambda I^*+\rho)(\mu+\alpha+\gamma-r)} \\ &\left(1 - \frac{1}{R_0}\left(\frac{\delta_1 R^*+\delta_2 V^*}{\lambda I^*}\right)\right), \frac{\rho(A+\delta_1 R^*+\delta_2 V^*)}{(\mu+\delta_2)(\mu-\lambda+\rho)}\left(\frac{1}{R_0}\right), \\ &\frac{\gamma\lambda(A+\delta_1 R^*+\delta_2 V^*)}{(\mu+\delta_1)(\mu-\lambda I^*+\rho)(\mu+\alpha+\gamma-r)}\left(1 - \frac{1}{R_0}\left(\frac{\delta_1 R^*+\delta_2 V}{\lambda I^*}\right)\right)\Bigg). \end{aligned} \tag{15}$$

2.4 Basic Reproduction Number (R_0)

The R_0 is obtained using the next generation operator matrix method [10]. In order to determine the number of individuals infected with disease arising when an infective is present in a host population of susceptible humans during his or her infectious lifetime, R_0 will be depending on

the epidemiological characteristics of the disease and the pattern of behavior of the population. If the duration last longer than expected, it will lead to a higher transmission of the disease which in turn increases the R_0.

Theorem 2.3 ([6], [18]). *Define* $X_s = \{x = 0 \mid x_i, i = 1,2,3,\ldots\}$. *In order to compute for* R_0, *we distinguished new infections from all other changes in the population.*
Let $F_i(x)$ *be the rate of new clinical manifestations of disease symptoms in compartment* i, *also, let* V_i^+ *be the rate at which individuals move into compartment* i *through other means and* V_i^- *be the rate at which individuals move out of compartment* i. *Then* $\dot{x}_i = f_i(x) = F_i(x) - V_i(x)$, $i = 1,2,3,\ldots$, *and* $V_i(x) = V_i^- - V_i^+$, *such that* F *is a non negative matrix and* V *is a non singular matrix.*

Proof.

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & \lambda\left(\frac{A}{\mu+\rho}\right) & 0 & 0 \\ 0 & \gamma & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{16}$$

and

$$V = \begin{pmatrix} (\mu+\rho) & 0 & \delta_1 & \delta_2 \\ 0 & (\mu+\alpha+\gamma)-r & 0 & 0 \\ 0 & 0 & (\mu+\delta_1) & 0 \\ \rho & 0 & 0 & (\mu+\delta_2) \end{pmatrix} \quad \text{and} \tag{17a}$$

$$V^{-1} = \begin{pmatrix} \frac{\mu+\delta_2}{\mu(\mu+\rho+\delta_2)} & 0 & -\frac{(\mu+\delta_2)\delta_1}{(\mu+\delta_1)\mu(\mu+\rho+\delta_2)} & -\frac{\delta_2}{\mu(\mu+\rho+\delta_2)} \\ 0 & \frac{1}{(\mu+\alpha+\gamma)-r} & 0 & 0 \\ 0 & 0 & \frac{1}{(\mu+\delta_1)} & 0 \\ -\frac{\mu+\delta}{\mu(\mu+\rho+\delta_2)} & 0 & \frac{\rho\delta_1}{(\mu+\delta)\mu(\mu+\rho+\delta_2)} & \frac{\mu+\rho}{\mu(\mu+\rho+\delta_2)} \end{pmatrix}. \tag{17b}$$

Therefore, R_0 is the largest eigenvalue of the spectral radius given by

$$R_0(FV^{-1}) = \frac{\lambda A}{(\mu+\rho)(\mu+\alpha+\gamma)-r}. \tag{18}$$

When $R_0 < 1$, the infections vanishes out of the host population. But if $R_0 > 1$, then the infections ravages and becomes endemic, which calls for appropriate medical interventions to stop the disease spread. □

3. Local and Global Stability Analysis of Infection Absent Steady State

3.1 Local Analysis

Theorem 3.1 ([2], [4]). *The infection free steady state* E^0 (14) *is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. The Jacobian matrix of (1) at infection free steady state solution (14) is given by

$$J(E^0)=\begin{pmatrix} -(\mu+\rho) & \lambda\left(\frac{A}{\mu+\rho}\right) & \delta_1 & \delta_2 \\ 0 & \lambda\left(\frac{A}{\mu+\rho}\right)-(\mu+\alpha+\gamma)-r & 0 & 0 \\ 0 & \gamma & -(\mu+\delta_1) & 0 \\ \rho & 0 & 0 & -(\mu+\delta_2). \end{pmatrix}. \tag{19}$$

From (19), the real parts are negative, but $\lambda\left(\frac{A}{\mu+\rho}\right)-(\mu+\alpha+\gamma)-r$ is positive. Then

$$\frac{\lambda A}{(\mu+\rho)(\mu+\alpha+\gamma)-r} > \frac{(\mu+\alpha+\gamma)-r}{(\mu+\alpha+\gamma)-r}. \tag{20}$$

From (20), it implies that

$$(R_0-1)>0,\ \ -R_0>-1,\ \ R_0<1. \tag{21}$$

Thus, from (21), the infection free steady state is locally asymptotically stable. □

3.2 Global Analysis

Theorem 3.2 ([4], [10])**.** *If* $R_0<1$*, the infection free steady state solutions of* (14) *is globally asymptotically stable in* Ω.

Proof. We consider the Lyapunov function candidate $H(S,I,V,R):\Re^4\to\Re^+$ defined as

$$H(S,I,R,V)=\eta I \quad \eta\geq 0. \tag{22}$$

Differentiating $H(S,I,R,V)$ with respect to time become

$$\dot{H}=\eta\dot{I}, \tag{23}$$

substituting the second state equation of (1) into (23) yields

$$\dot{H}=\eta(\lambda S-(\mu+\alpha+\gamma)-r)I\leq\eta\left(\frac{A\lambda}{\mu+\rho}-(\mu+\alpha+\gamma)-r\right)I. \tag{24}$$

Since $S\leq S^0=\frac{A}{\mu+\rho}$, taking $\eta=\frac{1}{(\mu+\alpha+\gamma)-r}$ implies that

$$\dot{H}=(R_0-1)I\leq 0 \tag{25}$$

from (25), $\dot{H}=0$, only when $I=0$. Then, $S\to\frac{A}{\mu+\rho}$ and $N\to\frac{A}{\mu}$ as $t\to\infty$, therefore,

$$\{(S,I,R,V)\in\Omega\mid\dot{H}\leq 0\} \tag{26}$$

is the singleton E^0. Hence from the La-Salle invariance principle [12], when $R_0<1$, the global stability of infection free steady state is globally asymptotically stable. □

4. Local and Global Stability Analysis of Infection Persistent Steady State

4.1 Local Analysis

Theorem 4.1 ([4], [10])**.** *The infection persistent steady state solution* E^* *of* (15) *is locally asymptotically stable if* $R_0>1$.

Proof. The Jacobian matrix of (1) at infection persistent steady state solutions is given by

$$J(E^*) = \begin{pmatrix} -(\mu+\rho)-\lambda I^* & \lambda S^* & \delta_1 & \delta_2 \\ \lambda I^* & \lambda S^* - (\mu+\alpha+\gamma) - r & 0 & 0 \\ 0 & \gamma & -(\mu+\delta_1) & 0 \\ \rho & 0 & 0 & -(\mu+\delta_2) \end{pmatrix}. \tag{27}$$

The quartic polynomial of (27) is yields

$$G_1\lambda^4 + G_2\lambda^3 + G_3\lambda^2 + G_4\lambda + G_5, \tag{28}$$

where

$$\begin{aligned}
G_1 &= \lambda(\mu+\lambda)(\mu+\lambda+\delta_1)(\mu+\rho+\lambda+\delta_2)S^* + (1-I^*), \\
G_2 &= ((4-3I^*)\mu + (1-I^*)\delta_1 + (1-I^*)\delta_2 + \rho + (1-I^*)\alpha + (1-I^*)r + (1-I^*)\gamma), \\
G_3 &= ((6-3I^*)\mu^2 + ((3-2I^*)\delta_1 + (3-2I^*)\delta_2 + 3\rho + (3-2I^*)\alpha + (3-2I^*)r + (3-2I^*)\gamma)\mu \\
&\quad + ((1-I^*)\delta_2 + \rho + (1-I^*)\alpha + (1-I)r + \gamma)\delta_1 - ((-1+I^*)\delta_2 - \rho)(r+\alpha+\gamma)) \\
&\quad ((4-I^*)\mu^3 + ((3-I^*)\delta_1 + (3-I^*)\delta_2 + 3\rho + (3-I^*)\alpha + (3-I^*)r + (3-I^*)\gamma)\mu^2 \\
&\quad + (((2-I^*)\delta_2 + 2\rho + (2-I^*)\alpha, \\
G_4 &= (2-I^*)r + 2\gamma)\delta_1 - ((-2+I^*)\delta_2 - 2\rho)(r+\alpha+\gamma))\mu - \delta_1(((-1+I^*)\alpha + \\
&\quad (-1+I^*)r - \gamma)\delta_2 - \rho(r+\alpha+\gamma))) \\
G_5 &= \mu(\mu+\delta_1)(\mu+\rho+\delta_2)(r+\mu+\alpha+\gamma)(1-R_0).
\end{aligned} \tag{29}$$

Using the Descartes rule of sign [15], the quartic polynomial has unique positive real roots, if and only if $G_1 > 0$, $G_2 > 0$, $G_3 > 0$, $G_4 > 0$ and $G_5 < 0$. Then the infection persistent steady state (S^*, I^*, R^*, V^*) is locally asymptotically stable. □

4.2 Global Analysis

Theorem 4.2 ([11]). *The infection persistent steady state solutions* (15) *is globally asymptotically stable in* Ω *if* $R_0 > 1$.

Proof. We Consider a Lyapunov function of the form

$$H : \{(S,I,R,V) \in \Omega : S,I,R,V > 0\} \to \Re^{+4}, \tag{30}$$

such that

$$H(S,I,R,V) = \frac{(S-S^*)^2}{2S^*} + \left(I - I^* - I^* \ln \frac{I}{I^*}\right) + \left(R - R^* - R^* \ln \frac{R}{R^*}\right) + \left(V - V^* - V^* \ln \frac{V}{V^*}\right). \tag{31}$$

The derivative of $H(S,I,R,V)$ along the solutions of (1) is given by

$$\dot{H}(S,I,R,V) = \frac{(S-S^*)}{S^*}\frac{dS}{dt} + \frac{(I-I^*)}{I}\frac{dI}{dt} + \frac{(R-R^*)}{R}\frac{dR}{dt} + \frac{(V-V^*)}{V}\frac{dV}{dt}. \tag{32}$$

From the first equation in (1),

$$A = \mu S^* + \lambda S^* I^* + \rho S^* - \delta_1 R^* - \delta_2 V^*. \tag{33}$$

Also, the second equation in (1) yields,

$$(\mu+\alpha+\gamma) - r = \lambda S^*. \tag{34}$$

The third equation in (1) yields

$$(\mu+\delta_1)=\frac{\gamma I^*}{R^*}, \tag{35}$$

while the fourth equation in (1) yields

$$(\mu+\delta_2)=\frac{\rho S^*}{V^*}. \tag{36}$$

Substituting the above equations into (31) with some simplifications, we obtain

$$\begin{aligned}\dot{H}(S,I,R,V)=\frac{(S-S^*)}{S^*}\Big[&\mu(S-S^*)+\lambda(SI-S^*I^*)+\rho(S-S^*)+\delta_1(R-R^*)\\&+\frac{(R-R^*)}{R}\gamma\Big(\frac{I}{R}-\frac{I^*}{R^*}\Big)+\frac{(V-V^*)}{V}\rho\Big(\frac{S}{V}-\frac{S^*}{V^*}\Big)\Big].\end{aligned} \tag{37}$$

Note that,

$$SI-S^*I^*=S^*(I-I^*)+I(S-S^*) \tag{38}$$

and

$$\gamma\Big(\frac{I}{R}-\frac{I^*}{R^*}\Big)=\gamma I^*\Big[\frac{I}{I^*}-\frac{R}{R^*}-\frac{R^*I}{RI}+1\Big] \tag{39}$$

and

$$\rho\Big(\frac{S}{V}-\frac{S^*}{V^*}\Big)=\rho S^*\Big[\frac{S}{S^*}-\frac{V}{V^*}-\frac{V^*S}{VS^*}+1\Big] \tag{40}$$

Thus, (32) yields

$$\begin{aligned}\dot{H}(S,I,R,V)=\frac{(S-S^*)}{S^*}&[\mu(S-S^*)]+\lambda[S^*(I-I^*)]+I[S-S^*]+\rho(S-S^*)\\&+\delta_1(R-R^*)+\delta_2(V-V^*)+\frac{(I-I^*)}{I^*}[\lambda S^*-r]+\gamma I^*\Big[\frac{I}{I^*}-\frac{R}{R^*}-\frac{R^*I}{RI^*}+1\Big]\\&+\gamma I^*\Big[\frac{R}{R^*}-\frac{I}{I^*}-\frac{I^*R}{R^*I}+1\Big]+\rho S^*\Big[\frac{S}{S^*}-\frac{V}{V^*}-\frac{V^*S}{VS^*}+1\Big]\\&+\rho S^*\Big[\frac{V}{V^*}-\frac{S}{S^*}-\frac{S^*V}{V^*S}+1\Big]\end{aligned} \tag{41}$$

and

$$(\mu+\lambda I+\rho)\frac{(S-S^*)^3}{S^*}+\delta_1(R-R^*)+\delta_2(V-V^*)+[\lambda S^*-rI^*] \tag{42}$$

$$+2\gamma I^*\Big[2-\frac{R^*I}{RI^*}-\frac{I^*R}{R^*I}\Big]+2\rho S^*\Big[2-\frac{V^*S}{VS^*}-\frac{S^*V}{V^*S}\Big]. \tag{43}$$

Hence, for all $S,I,R,V>0$, $\dot{H}(S,I,R,V)\le 0$ holds when $S=S^*$, $I=I^*$, $R=R^*$, $V=V^*$, $R^*I=RI^*$, $V^*S=VS^*$. This clearly showed that the infection persistent steady state E^* is the singleton and the largest invariant set in

$$\{(S,I,R,V)\in\Omega:\dot{H}(S,I,R,V)=0\}. \tag{44}$$

By the La-Salle's invariant principle [11], trajectories of (1) with its initial data in $\Omega\to E^*$ as $t\to\infty$, imply that the infection persistent steady state E^* is globally asymptotically stable in Ω if $R_0>1$. □

5. Numerical Simulations

Table 1. Variables in Model (1) and their Meanings

Variable	Descriptions	Values	Source
$S(0)$	Susceptible individuals	0.95	Estimated
$I(0)$	Infected individuals	0.30	Estimated
$R(0)$	Recovered individuals	0.05	Estimated
$V(0)$	Vaccinated Individuals	0.10	Estimated

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

Parameters	Descriptions	Values	Source
A	Per Capita Recruitment Rate	500	Assumed
μ	Natural death rate	0.112	Assumed
ρ	Rate of vaccination of susceptible individuals	0.21	Assumed
δ_1	Rate of immunity loss	0.21	Assumed
δ_2	Rate at which vaccine wanes	0.34	Assumed
α	Disease induced death rate	0.0125	Assumed
γ	recovery rate due to other means	0.11	Assumed
r	Treatment rate	0.016	Assumed

Figure 1 describes the transmission profile of the susceptible individuals whose immunity is lost due to the waning of vaccines being administered to them. The decline in the profile shows that more susceptible individuals will become exposed or infected as a result of loss of immunity in their system.

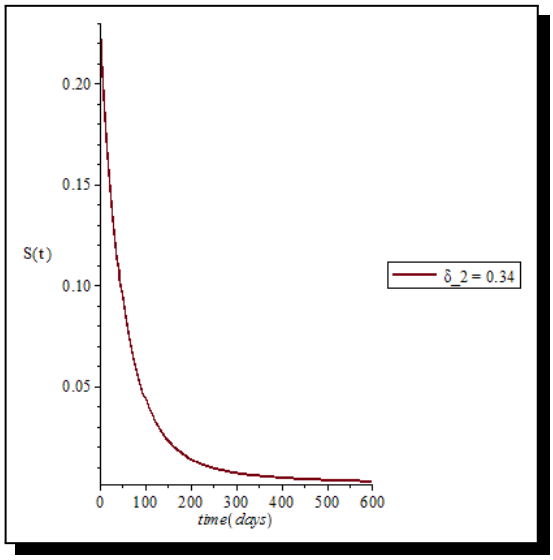

Figure 1. $S(t)$ against Time (t) varying δ_2

Figure 2 shows the level of treatment of infected individuals by varying $r = 0.016 - 0.119$. Infected individuals becomes recovered when proper treatment is applied to curtail the disease spread.

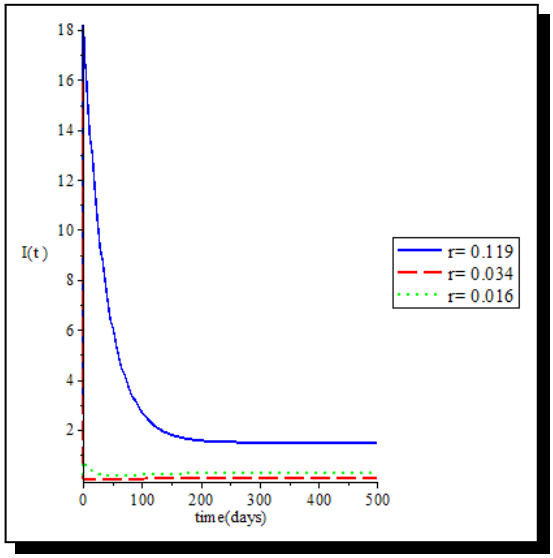

Figure 2. $I(t)$ against Time (t) varying r

Figure 3 The gradual rise in the disease profile of infected compartment state variable, shows that at in the absence of treatment and vaccination, the disease becomes a full blown epidemic in the human host population thereby leading to mortality.

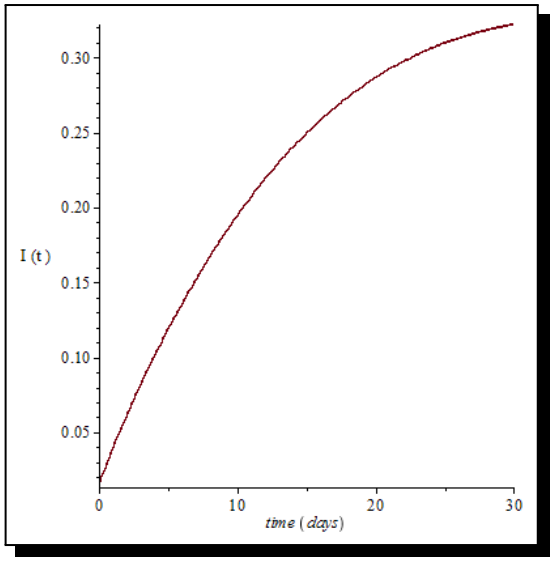

Figure 3. $I(t)$ against Time (t)

Figure 4 Describes the state variable compartment of the susceptible individual who are prone to contacting the disease in the presence of infected individual and the absence of intervention strategies in the human host population.

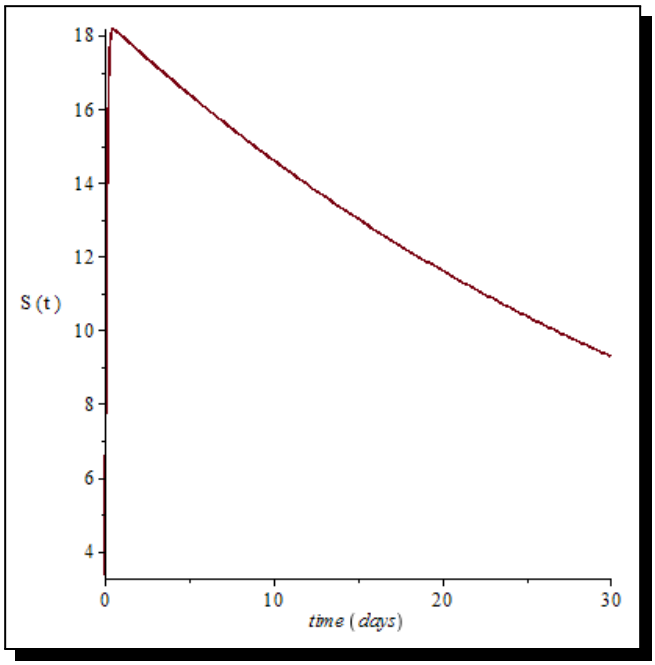

Figure 4. $S(t)$ against Time (t)

Figure 5 Illustrates the phase diagram of the interactions between the susceptible and the infected compartment.

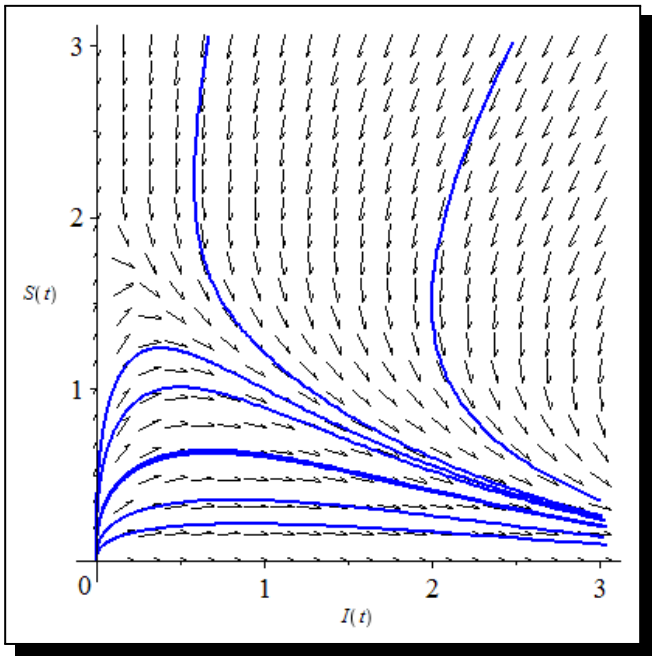

Figure 5. Phase diagram of $S(t)$ against $I(t)$

Figure 6 Describes the rate at which more susceptible individuals are given vaccinations at $\rho = 0.61 - 021$. When more susceptible individuals are given vaccinations, a level herd immunity is established, leading to the reduction and elimination of the disease.

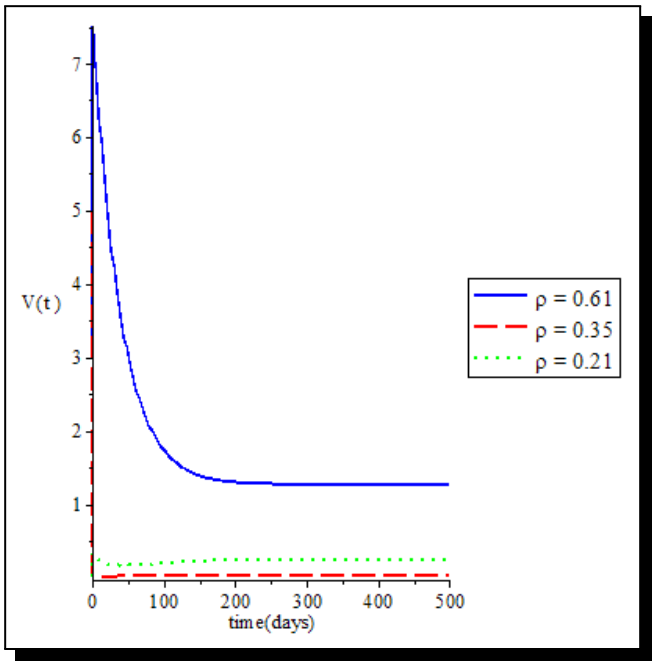

Figure 6. $S(t)$ against Time (t) varying ρ

Figure 7 Describes the gradual decline of infection, leading to a state of well-being in the compartmental state variable of the recovered class.

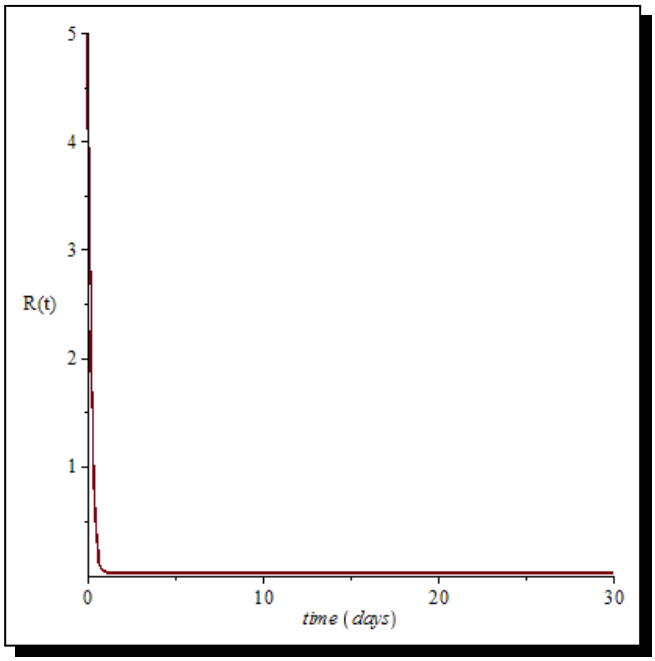

Figure 7. $R(t)$ against Time (t)

Figure 8 Shows the state variable of the vaccinated class. The more the susceptible individuals receives vaccinations, the infections gets eliminated in the human host population.

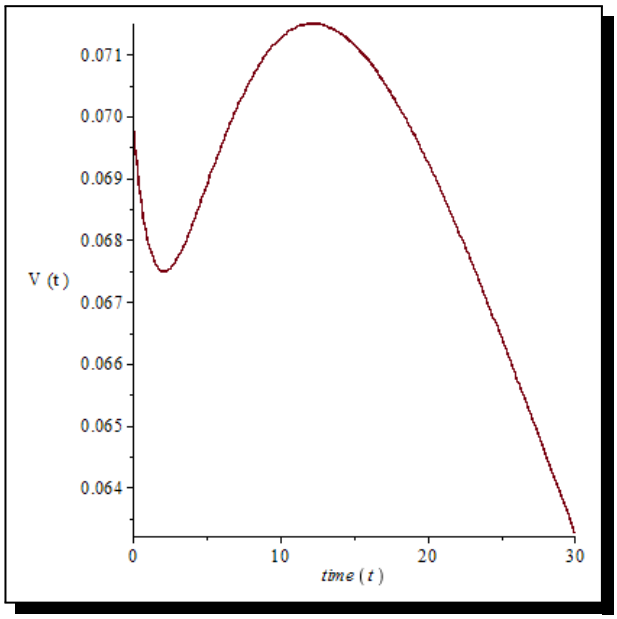

Figure 8. $V(t)$ against Time (t)

6. Conclusion

In this paper, we studied a deterministic SIRV model with non linear force of infection and treatment. It was shown that the solutions of the model exist, it is unique and positive as regards epidemic transmission and compartmental interactions in human host population. An invariant region where the model is mathematically well posed and realistic in an epidemic sense is investigated. However, the basic reproduction number (R_0) is determined and the stability of the model system at their steady state solutions is analyzed. It was shown that if $R_0 < 1$, infections leave the system, and if $R_0 > 1$, infections persists in the system. Lyapunov techniques were derived to analyze the model, and it was investigated that the model is locally and globally asymptotically stable. Also, vaccination and treatment parameters through simulations played important roles as an intervention strategy to achieve an infection free population. This work can be extended to incorporate environmental factors such as seasonality, age structure, optimal control.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

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