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Global Stability Analysis of a Mathematical Model on the Transmission Dynamics of Covid-19 with Vaccination

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ABSTRACT
In this work, we investigate the Global Stability *of a Mathematical model that describes the impact of vaccination on the dynamics of COVID-19 disease transmission in a human population.* The model, represented by a system of ordinary differential equations explains how infection from an index case, which could potentially lead to endemic state, can be averted through effective vaccination. The global stability analysis shows that, the diseases free state is *globally asymptotically stable*, when the basic reproduction number, $R_0 < 1$ in the absence of disease associated death. *This is supported by numerical simulation which suggests the combination of vaccination and non-pharmaceutical measures in the disease control.* We also show numerically that the disease invades when $R_0 > 1$ and that there is a transcritical bifurcation at $R_0 = 1$.

KEYWORDS: global stability, Covid-19; transcritical; vaccination; pharmaceutical, symptomatic; asymptomatic; bifurcation.

1. INTRODUCTION

SARS-CoV-2 known as COVID-19 is a human infectious coronavirus that originated in Wuhan, China, and has caused rapid spreading in China and around the world since December 2019 [1, 2], believed to have a zoonotic origin [3, 4, 5] and was identified and named by the World Health Organization (WHO) on January 10, 2020 following an ealier virus borne infection episode in Wuhan, China in December, 2019 [6]. The COVID-19 pandemic is considered as the biggest global threat because of thousands of confirmed infections, accompanied by thousands of deaths over the world [7]. Globally, as at 26th November 2021, there have been 259,502,031 confirmed cases of COVID-19, with 5,183,003 disease related deaths [8]. Mathematical modeling has become a powerful and important tool to understand infectious Disease dynamics and to improve on the control of the disease in a population. These models are often described by various forms such as: *SI*, *SIS*, *SIR*, or *SIRS*, etc. models, where *S* stands for susceptible subpopulation, *I* is infected subpopulation, and *R* is recovered Subpopulation. Depending on the mode of transmission of the disease under consideration, modification can be made to the above forms to give a detail explanation of the dynamics of the disease. The concept of Symptomatic, Asymptomatic and Surface Virus as considered in [6, 9], Vaccination in [9, 10], Isolation and Hospitalization in [11] and convalescence in [12] are modifications of the above general infectious disease models. Nonlinear ordinary differential equations have been used to explore the complex mechanisms of the dynamics of various systems in multidisciplinary fields: for instance, they are used in economics [13], quantum physics [14], chaos [15], medicine [16] and health diseases [17]. These models aim at optimizing predictive control of the parameters influencing the system dynamics. A Bat – Reservoir population transmission model was proposed in [5], to understand and simulate potential transmission from zoonotic source to humans. They estimated the basic reproductive number (R_0) as 2.4829. This value differs from 3.58, being the value estimated in [3]. The work of [2] suggests isolation and lockdown as a means of control of Covid-19 pandemic whereas some SIR models on Covid-19 have been proposed and carefully analyzed in [3, 5, 18, 19]. Lotka-Volterra based models of COVID-19 have been proposed and analyzed in [20].

In the work of [2], an epidemiological compartmental model that takes into account a super-spreading phenomenon of some individuals including fatality and hospitalized classes was proposed. The sensitivity analysis of their model shows that the most sensitive parameters to the basic reproduction number are infection

rate of humans, the rate at which exposed humans become infectious and the disease related death rate. Increase in the infection rate and the rate at which exposed individuals become infectious increase the basic reproduction number, and in contrast, the disease related death rate and the basic reproduction number are inversely related.

In the work of [6], the authors noted that, Covid-19 pandemic ravaging the world currently, will not end soon, as the result of their work shows damping oscillations. They aver that vaccination could be a possible remedy. Vaccination is an important public health control strategies that help to minimize the burden of an infectious disease spread and to delay a possible outbreak. Vaccination has the role of preventing healthy people from getting infected by a disease [21]. Various vaccination policies were studied in different mathematical models [9- 10, 22-26].

Various methods have been used in [17-18, 32-35] to show conditions of global asymptotic stability of infectious disease models. In this paper we extended an earlier work in [9] by investigating the global stability of the model exploring the technique in [17-18]. The total human population, $N(t)$, is divided into 6 classes namely, Susceptible class, $Q(t)$, Latent class L(t), Symptomatic class, $S(t)$, Asymptomatic class $A(t)$, Recovered class $R(t)$ and Vaccinated class V(t)). State variables in the model are given in Table 1 and the movement between compartments is summarized in Figure 1, the individual pathways to be discussed below.

1. List of model variable

State Variables	**Description**
N	Total Human Population.
L	Latent or Exposed non-infectious Human Population
Q	Susceptible Human Population
S	Symptomatic infectious Human Population
A	Asymptomatic infectious Human Population.
R	Recovered but Susceptible Human Population
V	Vaccinated human Population
P	Number of Viruses on Surfaces
t	time

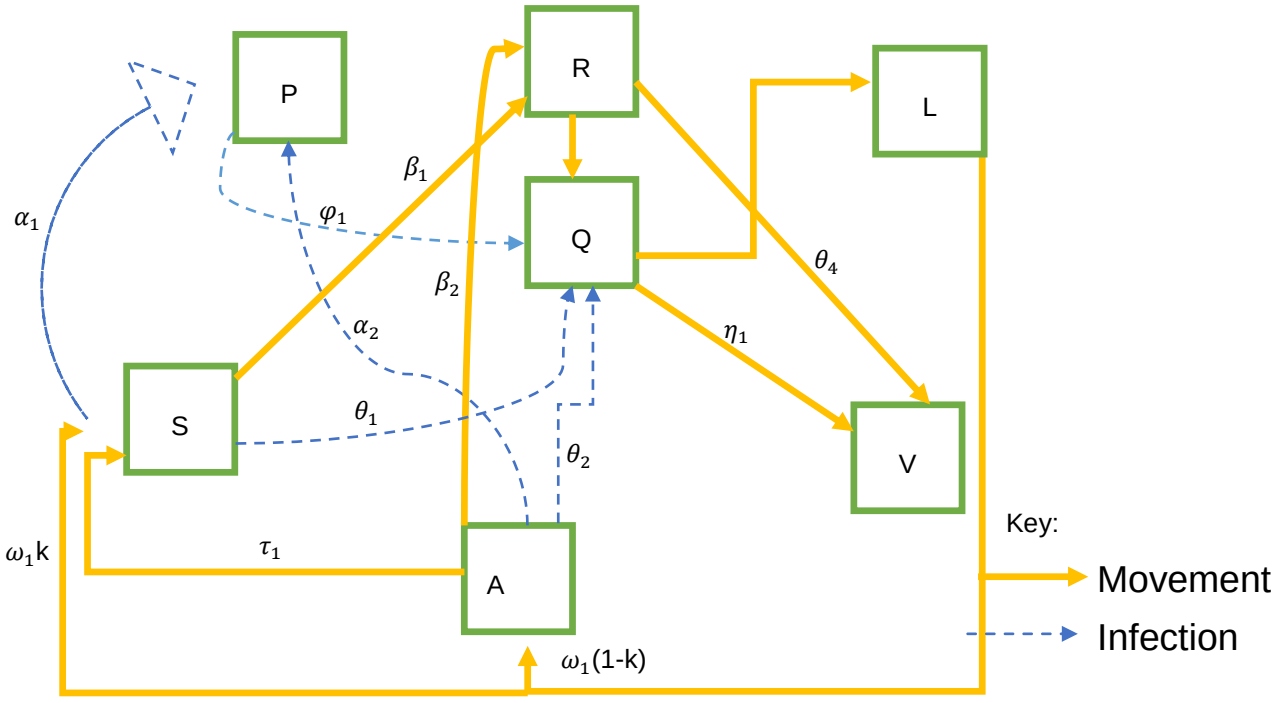


Fig. 1. Pathway diagram of the COVID-19 model showing (a) the progression (solid) and transmission (dashed) of the disease between compartments; the variable names are listed in Table 1. The connecting arrows are labelled with the associated rate constants, where the natural death of each of the classes are not shown for clarity.

2: THE MODEL

The entire human population is described by the equation

$$N = Q + L + S + A + \mathrm{V} + R \qquad (2.1)$$

Susceptible humans get infected by either contacting symptomatic and asymptomatic humans or viruses from surfaces at a rates $\theta_1 \frac{S}{N} Q, \theta_2 \frac{A}{N} Q$ and $\varphi_1 VQ$ respectively, where, θ_1, θ_2 and φ_1 are rate constants. The fractions $\frac{S}{N}$ *and* $\frac{A}{N}$ are the probabilities that the contacts are with symptomatic and asymptomatic humans. We note that humans in class E are in the exposed stage of infection and are not infectious. Susceptible humans are recruited into the population through a constant birth rate, λ_1 with a correction term $\theta_3 N^2$, stopping the population from growing without limit in the absence of the disease, where θ_3 is per capita resource availability for the human population. Exposed humans become infectious at a rate, $\omega_1 L$ in which a proportion, k of them become asymptomatic. This assumption is different from that of [5], where they suggested two incubation period even though they meant a single incubation period. All human classes die naturally at per capita rate, μ_1 while some individuals in the S class die at an additional rate $\sigma_1 S$ from the disease. We also assume that recovered Covid-19 patients are recruited into the vaccinated class at a rate, $\theta_4 R$ or, become susceptible again at a rate $\rho_1 R$ with θ_4 and ρ_1 as rate constants. Surface virus dies at rate $\gamma_1 P$ while symptomatic and asymptomatic humans contribute to the emergence of surface viruses at rates $\alpha_1 S$ and $\alpha_2 A$ respectively with α_1 and α_2 as rate constant. Susceptible Humans are vaccinated at a rate $\eta_1(1-m)Q$, where η_1 as rate constant and m is the proportion of people unwilling to be vaccinated. We assume that vaccinated humans become susceptible at a rate $\eta_2 V$ as the effectiveness of the vaccine wears out. The proposed model consistent with the above assumptions is given as:

$$\frac{dQ}{dt} = \lambda_1 N + \rho_1 R + \eta_2 V - \left(\theta_1 \frac{S}{N} + \theta_2 \frac{A}{N} + \varphi_1 P + \eta_1(1-m) + \mu_1\right) Q - \theta_3 N^2 \quad (2.2)$$

$$\frac{dL}{dt} = \left(\theta_1 \frac{S}{N} + \theta_2 \frac{A}{N} + \varphi_1 P\right) Q - (\omega_1 + \mu_1) L \quad (2.3)$$

$$\frac{dS}{dt} = (1-k)\omega_1 L + \tau_1 A - (\beta_1 + \sigma_1 + \mu_1) S \quad (2.4)$$

$$\frac{dA}{dt} = k\omega_1 L - (\beta_2 + \tau_1 + \mu_1) A \quad (2.5)$$

$$\frac{dV}{dt} = \eta_1(1-m)Q + \theta_4 R - (\eta_2 + \mu_1) V \quad (2.6)$$

$$\frac{dR}{dt} = \beta_1 S + \beta_2 A - (\theta_4 + \rho_1 + \mu_1) R \quad (2.7)$$

$$\frac{dP}{dt} = \alpha_1 S + \alpha_2 A - \varphi_2 PQ - \gamma_I P \quad (2.8)$$

$$\frac{dN}{dt} = (\lambda_1 - \mu_1) N - \sigma_1 S - \theta_3 N^2 \quad (2.9)$$

Equation (2.9) is obtained by adding equations (2.2)-(2.7)

2.1 Parameter Values and Nondimensionalisation

All the model parameters are listed in Table 2 below together with values taken from various sources.

Table 2. List of model parameters.

Symbols	Description	Value	Units	Source
λ_1	Per capita birth rate	0.0000433	Day^{-1}	[5,6,9]
θ_1	Infectious rate between Susceptible and symptomatic human population	0.05	Day^{-1}	[5,6,9]
θ_2	Infectious rate between Susceptible and Asymptomatic human population	0.124	Day^{-1}	[5,6,9]
φ_1	Infectious rate between surface virus and Susceptible Human population	0.00000123	$Virus^{-1}Day^{-1}$	[5,6,9]
β_1	Recovery rate of Symptomatic human population.	0.0987	Day^{-1}	[5,6,9]
β_2	Recovery rate of Asymptomatic human population	0.854	Day^{-1}	[15,6,9]
θ_3	Per capita Resources available for the human Population	0.00024	$Human^{-1}Day^{-1}$	[5,6,9]

μ_1	Natural death rate	0.0000357	Day^{-1}	[5,6,9]
τ_1	Rate of loss of Asymptomatic status	0.035	Day^{-1}	[5,6,9]
k	Proportion of exposed Human becoming infectious	0.005	Non-dimensional	[5,6,9]
α_1	Contribution of Symptomatic humans to surface Viruses.	0.0398	Viruses$Human^{-1}Day^{-1}$	[5,6,9]
α_2	Contribution of Asymptomatic humans to surface viruses	0.001	Viruses$Human^{-1}Day^{-1}$	[5,6,9]
φ_2	Depletion rate of surface virus due to contact with Susceptible Human	0.00000123	$Virus^{-1}Day^{-1}$	[5,6,9]
ω_1	Transition rate from exposed State to infectious State	0.000479	Day^{-1}	[5,6,9]
γ_1	Mortality rate of virus on surfaces	0.01	Day^{-1}	[5,6,9]
σ_1	Disease induced death rate	0.043	Day^{-1}	[11]
η_1	Rate at which susceptive humans are vaccinated.	0.0196	$Human^{-1}Day^{-1}$	Calculated from [6]
ρ_1	Rate at which recovered humans are been susceptible	0.084	Day^{-1}	assumed
m	Proportion of humans that were not vaccinated as a result of Conspiracy theory.	0.98	Day^{-1}	Calculated from [4]
η_2	Vaccine wearing out rate	0.08	Day^{-1}	[9]
θ_4	Rate of vaccination of recovered humans	0.89	Day^{-1}	[9]

We define nondimensional variables of the form:

$$\hat{Q} = \frac{Q}{N}, \hat{L} = \frac{L}{N}, \hat{S} = \frac{S}{N}, \hat{A} = \frac{A}{N}, V = \frac{V}{N}, \hat{R} = \frac{R}{N}, \hat{P} = \frac{P}{P_0}, \hat{N} = \frac{N}{N_0}, \hat{t} = \frac{t}{t_0}, \quad (2.10)$$

such that

$$\hat{Q} + \hat{L} + \hat{S} + \hat{A} + \hat{V} + \hat{R} = 1 \quad (2.11)$$

We substitute (2.10) in (2.2) – (2.9), carry out some algebraic simplifications and rescale time with the rate of Vaccinated human population. By defining the following dimensionless parameters.

$$t_0 = \frac{1}{\eta_1}, \lambda = \frac{\lambda_1}{\eta_1}, \rho = \frac{\rho_1}{\eta_1} \quad a = \frac{\theta_1}{\eta_1}, b = \frac{\theta_2}{\eta_1}, \mu = \frac{\mu_1}{\eta_1}, d = \frac{\varphi_1 V_0}{\eta_1}, e = \frac{\beta_2}{\eta_1}, \sigma = \frac{\sigma_1}{\eta_1},$$

$$f = \frac{\theta_3 N_0}{\eta_1}, \tau = \frac{\tau_1}{\eta_1}, \omega = \frac{\omega_1}{\eta_1}, g = \frac{\alpha_1 N_0}{\eta_1 V_0}, h = \frac{\alpha_2 N_0}{\eta_1 V_0}, \theta = \frac{\theta_4}{\eta_1}, \gamma = \frac{\gamma_1}{\eta_1}, \eta = \frac{\eta_2}{\eta_1}, \varphi = \frac{\varphi_2 N_0}{\eta_1} \quad (2.12)$$

$$\beta = \frac{\beta_1}{\eta_1},$$

and dropping the hats for notational simplicity, we obtain the nondimensional system;

$$\frac{dQ}{dt} = \lambda(1-Q) + \rho R + \eta V - [aS + bA + dP + (1-m)]Q + f(Q-1)N + \sigma QS, \quad (2.13)$$

$$\frac{dL}{dt} = (aS + bA + dP)Q - (\omega + \lambda)L + fLN + \sigma LS, \quad (2.14)$$

$$\frac{dS}{dt} = (1-k)\omega L + \tau A - (\beta + \sigma + \lambda)S + fSN + \sigma S^2, \quad (2.15)$$

$$\frac{dA}{dt} = k\omega L - (e + \tau + \lambda)A + fAN + \sigma AS, \quad (2.16)$$

$$\frac{dV}{dt} = (1-m)Q + \theta R - (\eta + \lambda)V + fVN + \sigma VS, \quad (2.17)$$

$$\frac{dR}{dt} = \beta S + eA - (\theta + \rho + \lambda)R + fRN + \sigma RS, \quad (2.18)$$

$$\frac{dP}{dt} = gSN + hAN - \varphi NPQ - \gamma P, \quad (2.19)$$

$$\frac{dN}{dt} = (\lambda - \mu)N - \sigma SN - fN^2, \quad (2.20)$$

subject to the initial conditions,

$Q(0) = q_0, L(0) = 1 - q_0, S(0) = A(0) = \mathrm{V}(0) = \mathrm{R}(0) = \mathrm{P}(0) = 0$

3.0 MODEL ANALYSIS

3.1 The Basic Reproduction Number, R_0

Using the next generation matrix approach [27, 28, 30], we consider the equation

$W' = \frac{dW}{dt}$, where

$W' = FW - MW$

(3.1)

$$F = \begin{bmatrix} 0 & aQ_0 & bQ_0 & dQ_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad M = \begin{bmatrix} h_1 & 0 & 0 & 0 \\ -h_6 & h_2 & -\tau & 0 \\ -h_7 & 0 & h_3 & o \\ 0 & -h_4 & -h_5 & h_8 \end{bmatrix}, \quad W = \begin{bmatrix} L \\ S \\ A \\ P \end{bmatrix}$$

Here, FW represents the emergence of new infections, MW the transition of these infections among compartments and W, the reservoir of infection where,

$\lambda > \mu, \quad k < 1, \quad m < 1, \quad r_1 = \lambda - \mu, \quad r_2 = 1 - k, \; r_3 = 1 - m$

$h_1 = \omega + \mu, \; h_2 = \beta + \sigma + \mu, \; h_3 = e + \tau + \mu,$

(3.2)

$h_4 = \frac{gr_1}{f}, h_5 = \frac{hr_1}{f}, h_6 = r_2\omega, \; h_7 = k\omega, \; h_8 = \frac{\varphi r_1}{f} + \gamma$

The largest eigenvalue of $G = FM^{-1}$ is the basic reproduction number.

$$G = \frac{1}{g_0}\begin{bmatrix} g_9 & 0 & 0 & 0 \\ K_2 & g_8 & K_6 & 0 \\ K_3 & 0 & K_7 & 0 \\ K_4 & K_5 & K_8 & B_8 \end{bmatrix},$$

(3.3)

Where $g_8 = h_1h_3h_4, \; g_9 = h_2h_3h_8, \; K_2 = h_3h_6h_8 + \tau h_7h_8, K_3 = h_2h_7h_8$

$K_4 = h_2h_5h_7 + h_3h_4h_6 + \tau h_4h_7, \; K_5 = h_1h_3h_4$

$K_6 = \tau h_1h_8, \; K_7 = h_1h_2h_8, \; K_8 = h_1(h_2h_5 + \tau h_4), B_7 = h_1h_2, B_8 = B_7h_3$

The highest eigenvalue of G gives the basic reproduction number:

$R_0 = \frac{\omega Q_0\{L_1(e+\tau+\mu)+L_2(\beta+\sigma+\mu)\}}{(\omega+\mu)(e+\tau+\mu)(\beta+\sigma+\mu)(f\sigma+\theta r_1)}$

(3.4)

Where, $L_1 = r_2\{af\sigma + r_1(a\theta + dg)\}, L_2 = k\{bf\sigma + r_1(b\theta + dh)\}$ and

$Q_0 = \frac{\eta+\mu}{\eta+\mu+r_2}$

3.2 Positivity, Existence and Uniqueness of Solution

The model is described in the domain

$\Omega \in \mathbb{R}^8 = \{Q, \; L, S, A, R, V, P, N: Q \geq 0, L \geq 0, S \geq 0, A \geq 0, R \geq 0, V \geq 0,$

$P \geq 0, N > 0, \quad Q + L + S + A + V + R = 1\}$ *(3.5)*

Assuming all variables are positive $at\ t = 0$, then $Q(0) + L(0) + S(0) + A(0) + V(0) + R(0) = 1$. If $L = 0$, and all other variables are in Ω, then, $\frac{dL}{dt} \geq 0$, this is also the case for variables in (2.15) - (2.19). If $N = 0, \; then, \; \frac{dN}{dt} = 0$. But if $N > 0$ and assuming $\lambda > \mu$, then with suitable initial conditions, $\frac{dN}{dt} > 0 \; \forall \; t > 0$. We observe that the right-hand side of (2.15) - (2.20)

is continuous with continuous partial derivatives. Thus, solutions exist and are unique and the model has mathematically and biologically relevant solutions in the domain $\Omega \; \forall \; t \in [0, \infty)$.

3.3 Steady State Solution

The equilibrium point is given as $E_0 = (Q, L, S, A, V, R, P) = (Q_0, 0,0,0, V_0, 0,0)$. Substituting S = 0 and A = 0 into the right hand side of (2.18), and (2.19) gives R = 0, and P = 0. Further substitution of the values of S, A, R, and P into (2.14) gives L=0. Using $S=A=R=L=P=0$ in (2.17) and (2.12) gives $V_0 = \frac{r_2}{\eta+\mu+r_2}$ and $Q_0 = \frac{\eta+\mu}{\eta+\mu+r_2}$ respectively. At the disease free state, all humans are entirely susceptible and we obtain from (2.20) the following logistic equation,

$\frac{dH}{dt} = r_1 N - fN^2$

(3.6)

With solution

$N(t) = \frac{KN_O}{N_O+(K-N_O)e^{-r_1 t}}$,

(3.7)

where r_1 is as defined above and $K = \frac{r_1}{f}$. As $t \to \infty, N(t) \to K,$ which is the carrying capacity of the environment.

3.4 Local Stability Analysis of the Disease Free Equilibrium (E_0)

Lemma 3.1: The disease free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

The proof of Lemma 3.1 is given in [9]**.**

3.5 Global Stability Analysis of the Disease Free Equilibrium (E_0)

Following the method used in [18-19], we consider the following two conditions, H_1 and H_2.

(H_1): For $\frac{dX}{dt} = F(X, 0)$, E_0 is globally asymptotically stable (g.a.s.),

(H_2): $\hat{G}(X, Y) = AY - G(X, Y) \geq 0 \; \forall \; (X, Y) \in \Omega$

Lemma 3.2. The disease free equilibrium of the model equations (2.13) to (2.19) is globally asymptotically stable (g.a.s) if the system is locally asymptotically stable for R_0 <1 and in addition, $H_1$and H_2 hold.

Proof

We write the model equations (2.13) to (2.19) in the form

$$\frac{dX}{dt} = F(X, Y) = \begin{bmatrix} \lambda(1-Q) + \rho R + \eta V - [aS + bA + dP + (1-m)]Q + f(Q-1)N + \sigma QS \\ (1-m)Q + \theta R - (\eta + \lambda)V + fVN + \sigma VS \\ \beta S + eA - (\theta + \rho + \lambda)R + fRN + \sigma RS \end{bmatrix}$$

$$\frac{dY}{dt} = G(X, Y) = \begin{bmatrix} (aS + bA + dP)Q - (\omega + \lambda)L + fLN + \sigma LS \\ (1-k)\omega L + \tau A - (\beta + \sigma + \lambda)S + fSN + \sigma S^2 \\ k\omega L - (e + \tau + \lambda)A + fAN + \sigma AS \\ gSN + hAN - \varphi NPQ - \gamma P \end{bmatrix}$$

where $X = (Q, V, R)$ and $Y = (L, S, A, P)$, with the components of $X \in R^3$, denoting uninfected population and the components of $Y \in R^4$, denoting the infected population.

From Section *3.3*, $E_0 = (Q_0, 0,0,0, V_0, 0,0)$. Now,

$$F(X, 0) = \begin{bmatrix} \eta V + \mu - (r_3 + \mu)Q \\ r_3 Q - (\eta + \mu)V \\ 0 \end{bmatrix} \qquad 3.13$$

From *(3.13);*

$\frac{dV}{dt} = \frac{r_3(\eta + \mu)}{\eta + \mu + r_2} - (\eta + \mu)V$

$\frac{dV}{dt} + (\eta + \mu)V = \frac{r_3(\eta+\mu)}{\eta+\mu+r_2}$ *3.14*

Integrating factor (IF) of (3.14) $= e^{\int(\eta+\mu)dt} = e^{(\eta+\mu)t}$, then;

$V.e^{(\eta+\mu)t} = \int e^{(\eta+\mu)t}\left(\frac{r_3(\eta+\mu)}{\eta+\mu+r_2}\right)dt$

$V.e^{(\eta+\mu)t} = \left(\frac{r_3}{\eta+\mu+r_3}\right)e^{(\eta+\mu)t} + K$, *3.15*

Where K is the constant of integration. Multiplying (3.15) through by $e^{-(\eta+\mu)t}$, then;

$$V(t) = \frac{r_3}{\eta+\mu+r_3} + Ke^{-(\eta+\mu)t}. \quad 3.16$$

At $t = 0,\ K = V(0) - \frac{r_3}{\eta+\mu+r_2}$. Thus, *(3.16)* becomes:

$$V(t) = \frac{r_3}{\eta+\mu+r_3} + A_0e^{-(\eta+\mu)t}. \quad 3.17$$

Where $A_0 = V(0) - \frac{r_3}{\eta+\mu+r_2}$. From *(3.17)*,

$V(t) = V_0\ as\ t \to \infty$.

Similarly, $Q(t) = \frac{r_3}{\eta+\mu+r_2}\ as\ t \to \infty$.

Hence, H1 holds.

$$\frac{dY}{dt} = G(X,Y) = \begin{bmatrix} (aS+bA+dP)Q - (\omega+\lambda)L + fLN + \sigma LS \\ (1-k)\omega L + \tau A - (\beta+\sigma+\lambda)S + fSN + \sigma S^2 \\ k\omega L - (e+\tau+\lambda)A + fAN + \sigma AS \\ gSN + hAN - \varphi NPQ - \gamma P \end{bmatrix}$$

$$A = \begin{bmatrix} -(\omega+\mu) & aP & bP & dP \\ (1-k)\omega & -(\beta+\sigma+\mu) & \tau & 0 \\ k\omega & 0 & -(e+\tau+\mu) & 0 \\ 0 & gN & hN & -(\varphi NP-\gamma) \end{bmatrix}$$

$$AY = \begin{bmatrix} (aS+bA+dV)P - (\omega+\mu)L \\ (1-k)\omega L + \tau A - (\beta+\sigma+\mu)S \\ k\omega L - (e+\tau+\mu)A \\ gN + hN - (\varphi NP-\gamma)V \end{bmatrix}$$

$$\hat{G}(X,Y) = AY - G(X,Y)$$

$$= \begin{bmatrix} (aS+bA+dP)Q - (\omega+\mu)\mathrm{L} \\ (1-k)\omega L + \tau A - (\beta+\sigma+\mu)S \\ k\omega L - (e+\tau+\mu)A \\ gN + hN - (\varphi NQ-\gamma)P \end{bmatrix} - \begin{bmatrix} (aS+bA+dP)Q - (\omega+\lambda)L + fLN + \sigma LS \\ (1-k)\omega L + \tau A - (\beta+\sigma+\lambda)S + fSN + \sigma S^2 \\ k\omega L - (e+\tau+\lambda)A + fAN + \sigma AS \\ gSN + hAN - \varphi NPQ - \gamma P \end{bmatrix}$$

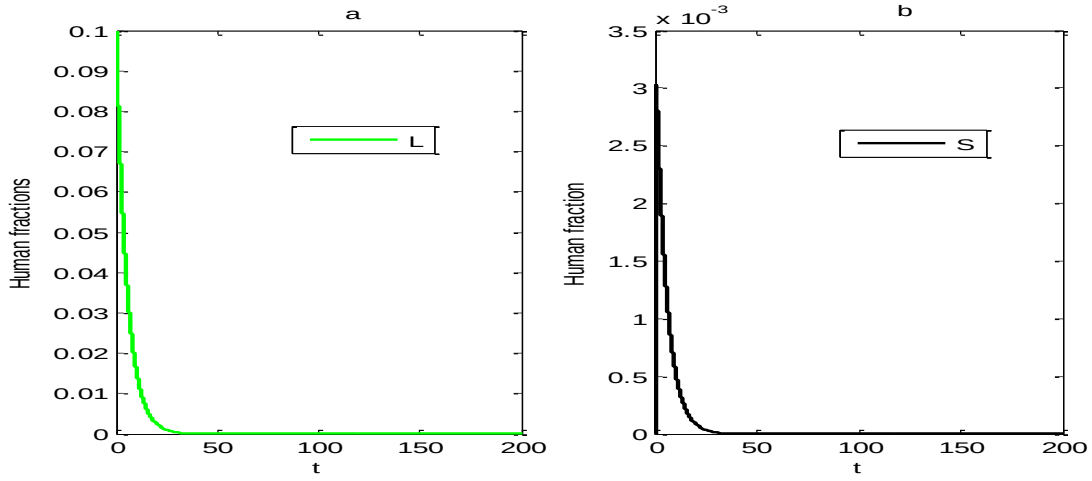
$$\therefore \hat{G}(X,Y) = \begin{bmatrix} -\sigma LS \\ -\sigma S^2 \\ -\sigma AS \\ 0 \end{bmatrix}$$

$\rightarrow \hat{G}(X,Y) \geq 0\ \forall\ (X,Y) \in \Omega\ iff\ \sigma = 0$. *It follows that* H_1 *and* H_2 *hold when* $\sigma = 0$.

Thus, E_0 is globally asymptotically stable, when $\sigma = 0$.

3.6 Numerical Solution

We carry out the numerical simulations with MATLAB's ODE15s, using the following valued dimensionless parameters :$\lambda = 0.0221, a = 2.551, b = 6.326, d = 0.0000628, \beta = 5.0357, e = 43.571, f = 0.01224, \mu = 0.00182, \eta = 4.081, \tau = 1.7857, g = 2.0306, h = 0.05102, \omega = 0.0244, C\rho = 4.286, k = 0.005, \theta = 0.42857, \varphi = 0.0000628, \gamma = 0.5102, m = 0.01$ with initial conditions Q = 0.9, L = 0.1, S = 0, A = 0, V = 0, R=0, P = 0, N =



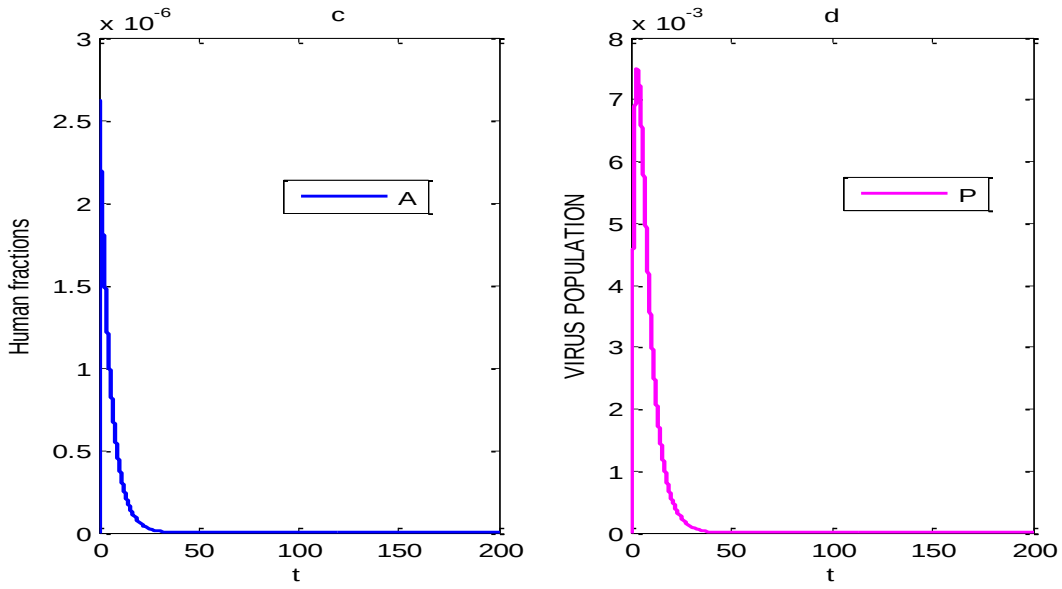


Fig.1. Results showing the effect of Vaccination on Symptomatic, Asymptomatic and Recovered humans and Surface Virus. where t = 1, represents approximately 5 days in real time. The initial conditions used are Q = 0.9, L = 0.1, S = 0, A = 0, V=0, R = 0, P = 0, N = 1 and the parameter values are given above.

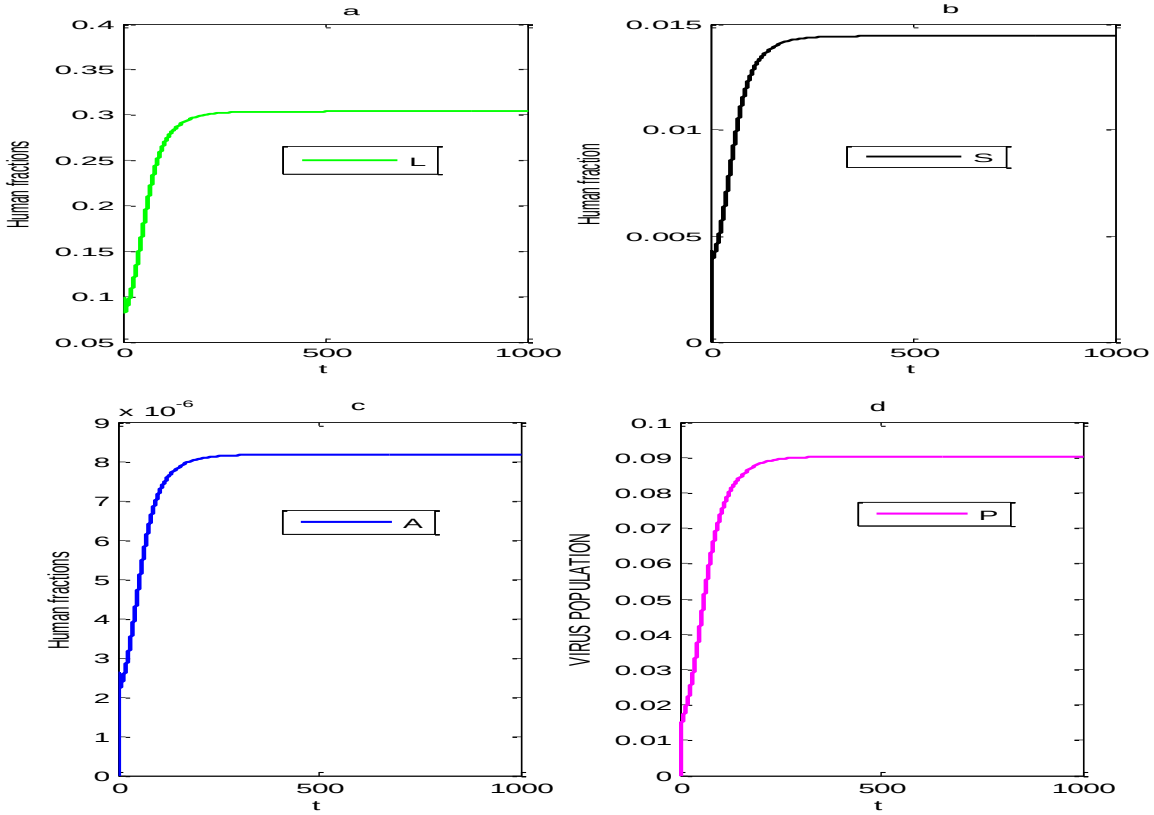


Fig.2. Result showing the effect of high contact rate between infectious and susceptible humans on the disease dynamics with $R_0 > 1$ and the values used for the simulations are the same as above with only a =3.25, b =4.126, d =0.728 and $\omega = 0.244, \sigma = 0.073, m = 0.99$

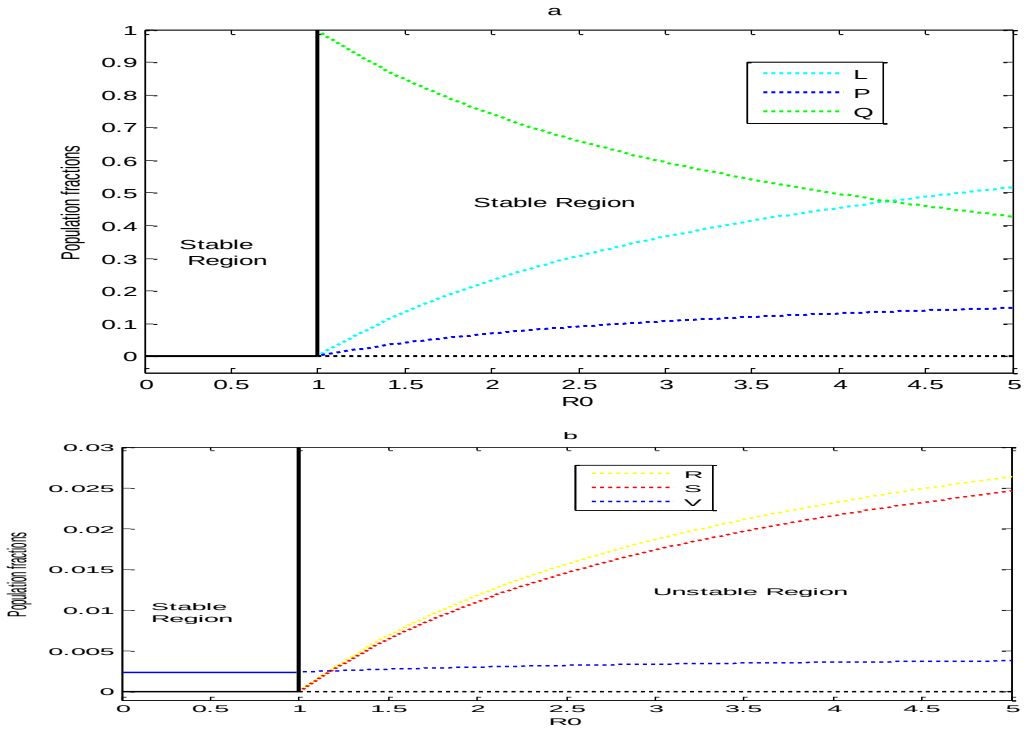


Fig.3. Results showing the disease free and endemic states for $R_0 < 1$ and $R_0 > 1$ respectively, as R_0 varies from 0 to 5 based on the parameter, d. The values used for the simulations are the same as those in Fig.2 except that d was varied upwards from zero.

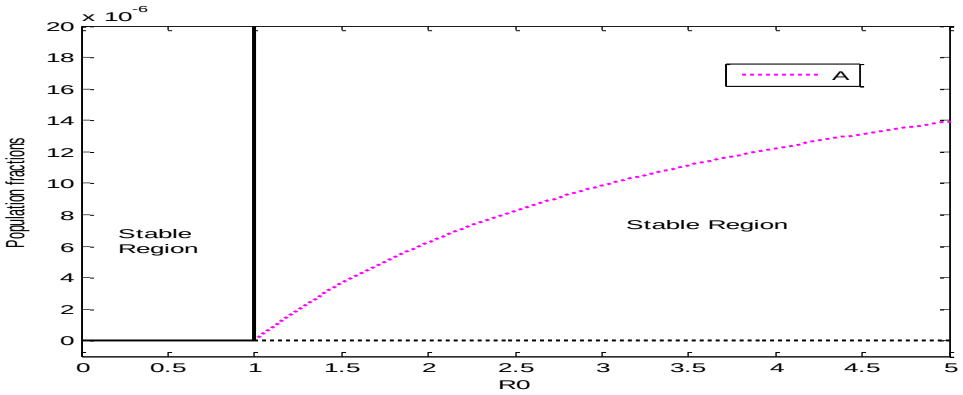


Fig.4. Results showing asymptomatic infection free and persistent states for $R_0 < 1$ and $R_0 > 1$ respectively, as R_0 varies from 0 to 5 based on the parameter, d. The values used for the simulations are the same as those in Fig.3.

3.5 Discussions

In this model, we describe the transmission of Covid-19 disease in an entirely susceptible human population due to the introduction of a single case and the effect of vaccination on the Disease dynamics. Using available data and with the introduction of vaccination, we obtain the Basic Reproduction Number, $R_0 = 0.28$ different from the results of [5], [6] and [9]. This value of R_0 suggests that the disease may likely die out due to vaccination as seen in fig.1a,b,c,d,; where the reservoir of infection comprising the latent or exposed, symptomatic and asymptomatic human populations and number of viruses in environmental decay,. This behavior is as a result of the impact vaccination in the system. This result agrees with the recommendation that was made in [6]. However, increasing the contact rates between infectious and susceptible humans will hinder the positive effect of vaccination. This agrees with the results of [9] and [10], which maintain that vaccination should be carried out in conjunction with other social measures that restrict contact rate between infectious and susceptible humans. The analysis shows that the disease free state is globally asymptotically stable in the absence of disease related death.

We note that some major parameters, a, b d and ω play significant roles in perpetuating the progression of the disease depending on whether they are high or low even though sensitivity analysis has not been carried out. Their high values will significantly increase R_0 despite the introduction of vaccination. If the transition rate from latent

period to infectiousness continues to increase leading to high number of symptomatic and asymptomatic humans and viruses in the environment without control, then there is every possibility that the disease will invade the population as shown in Fig2a,b,c, d where the level of infection, and viruses on environmental surfaces increase to a steady state, implying an endemic situation.. Thus, in other to eradicate the disease, control measures like Social distancing, contact tracing, testing, quarantine, treatment, etc. are to be considered alongside vaccination.

We use the parameter, d to vary R_0 where $d = 0.016$ corresponds to $R_0 = 1$. Fig.3a,b,c and Fig.4 show the relationship between R_0 and the disease compartments as it affects the entire population. The disease establishes itself for values of $R_0 > 1$ and dies out if $R_0 < 1$. Fig.3 and Fig.4 are bifurcation diagrams showing a switch from disease free state to disease persistent state. The result is obtained by plotting the steady states of the various compartments against different values of R_0. The plots show a transcritical bifurcation in the vicinity of $R_0 = 1$ as expected. Although we are not certain about whether or not the disease invades at $R_0 = 1$, but the disease free state is locally asymptotically stable for values of $R_0 < 1$ from the analysis of [9] and globally asymptotically stable with an additional condition that $\sigma = 0$. The disease free state becomes unstable when $R_0 > 1$, whereas, the endemic state becomes stable as expected.

4. CONCLUSION

In this work, we presented a mathematical model on the dynamics of Covid -19 disease with vaccination. The model focuses on the effect of vaccination in the transmission dynamics of Covid-19 in a totally susceptible population due to the introduction of an index case. Analysis of the model shows that with the introduction of Vaccination the disease will likely die out. However, control measures like Social distancing, contact tracing, testing, quarantine; treatment should be encouraged despite vaccination [9, 10], as there exists the possibility of the disease becoming endemic with vaccination alone. The numerical simulations show a transcritical bifurcation within the vicinity of $R_0 = 1$, and the model is globally asymptotically stable if $\sigma = 0$, in which case it carefully avoids disease related deaths to establish a status of global stability.

Conflict of Interest: There wass no conflict of interest

REFERENCES

1. B. Y. Ahmad, and H. Zeaid, (2020). COVID-19: Modeling, Prediction, and Control. Applied Sciences. *10*(11), *Appl. Sci. 10(11),3666;* https://doi.org/10.3390/app10113666
2. M. Y. Hyun, and P. L. Luis, (2021). Mathematical modeling of the transmission of SARS-CoV-Evaluating the impact of isolation in São Paulo State (Brazil) and lockdown in Spain associated with protective measures on the epidemic of Covid-19.
3. O. Amanso, N. Agueboh, P. U. Achimugwu, C. Okeke, B. E. Chukwuemeka, K. C. Nnamaga, E. Oshilim, (2020) Mathematical model of the early incidence and spread of COVID-19 in Nigeria combined with control measure. International Journal of Scientific and Engineering Research. 11(4):1110.
4. Y. Chayu, and W.Jin, (2020). A mathematical model for the novel coronavirus epidemic in Wuhan, China. Math Biosci Eng. 17(3):2708–2724. DOI: 10.3934/mbe.2020148
5. M. A. Khan, and A. Atangana, (2020). Modeling the dynamics of novel coronavirus (2019-nCov) with fractional derivative.Alexandria Eng. J; 59(4): Available:https://doi.org/10.1016/j.aej.2020.02.033
6. A. B. Okrinya, and E. Esekhaigbe, (2021). Mathematical modelling of the dynamics of COVID-19 disease transmission. Asian Research Journal of Mathematics.;17(1):123-137.
7. F. Ndarou, I. Area, J. Nieto, (2020). Torres D. Mathematical modeling of COVID-19 transmission dynamics with a case study of Wuhan. Chaos, Solitons, and Fractals. ;135(1046). Available:https://doi.org/10.1016/j.chaos.2020.109846.
8. Center for Disease Control and Prevention; 2019. Available:cdc.gov/coronavirus/2019-ncov/symptoms
9. A. B. Okrinya and C. N. Timinibife. (2021). The Impact of Vaccination on Covid-19 Disease Transmission Patterns in a Human Population: A Theoretical Analysis. Asian Research Journal of Mathematics.;17(1):123-137.
10. G. Nezihal, and K. Bilgen. Mathematical modelling of Covid-19 with the effect of vaccine AIP Conference Proceedings. 2021;2325:020065. Available: https://doi.org/10.1063/5.0040301
11. D. Wang, B. Hu, and C. Hu, (2020). Clinical characteristics of 138 hospitalized patients with 2019 Novel Corona virus infected Pneumonia in Wuhan, China, JAMA.;323(11). Available: https://jamanetwork.com
12. A. B. Okrinya, and J. I. Consul, (2019). Logistic mathematical model of Ebola virus disease with convalescence. International Journal of Applied Scientific and Research.;2(6):1-14.
13. F. Fatemeh, V. Karimi, C. O. Puig, (2021). Economic model predictive control of nonlinear systems using a linear parameter varying approach. International Journal of Robust and Nonlinear Control;.

14. C. Aoun, N. Boutabba, H. Eleuch, (2021). Alpha Model: A Mathematical Modeling Approach Applied to an Air Quality Monitoring Network Applied Mathematics & Information Sciences. 27 (1). DOI: 10.12785/amis/010104
15. A. Dokoumetzidis, A. Iliadis, P. Macheras, (2001). Nonlinear Dynamics and Chaos Theory: Concepts and Applications Relevant to Pharmacodynamics. 18:415-426.
16. D. S. Glass, X. Jin, I. H. Riedel-Kruse, (2021). Nonlinear delay differential equations and their application to modeling biological network motifs. 12(1788).
17. M. Rahman, and F. Gómez-Aguilarc, (2020). Investigating a nonlinear dynamical model of COVID-19 disease under fuzzy caputo, random and ABC fractional order derivative. 140:110232. doi: 10.1016/j.chaos.2020.110232
18. B. M. Ndiaye, and L. S. Tendeng, (2020). Analysis of the COVID-19 pandemic by SIR model and machine learning techniques for forecasting.
19. T. Chen, J. Rui, Q. Wang, Z. Zhao, J. Cui, L. Yin, (2020). A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. Infectious Diseases of Poverty. 9(24). Available: https//doi.0rg/10.1186/s40249-020-00640-3.
20. W. Wael, A. E. Mohammed, E. S. Aly, C. D. Matouk, S. A. Albosaily, E. M. Elabbasy, (2021). An analytical study of the dynamic behavior of Lotka-Volterra based models of COVID-19. 2021; 26(1).
21. A. Elazzouzi, A. Lamrani Alaoui, M. Tilioua, A. Tridane, (2019). Global stability analysis for a generalized delayed SIR model with vaccination and treatment. Adv Differ Equ. 2019;2019(1):532. Doi:10.1186/s13662-019-2447-z. Epub 2019 Dec 21. PMID: 32226453; pmcid: PMC7100696.
22. Y. Enatsu, (2012). Lyapunov functional techniques on the global stability of equilibria of SIS epidemic models with delays. Kyoto Univ. Res. Inf. Repos. 1792, 118–130.
23. Z. Lu, X. Chi, L. Chen, (2002). The effect of constant and pulse vaccination on SIR epidemic model with horizontal and vertical transmission. Math. Comput. Model. 36, 1039–1057.
24. Makinde, O. D.(2007). Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy. Appl. Math. Comput. 184(2), 842–848.
25. B. Shulgin, L. Stone, Z. Agur, (1998). Pulse vaccination strategy in the SIR epidemic model. Bull. Math. Biol. 60(6), 1123–1148.
26. Y. Ma, J. B. Liu, H. Li, (2018). Global dynamics of an SIQR model with vaccination and elimination hybrid strategies. Mathematics 6(12), 1–12.
27. N. Chitnis, (2005). Using mathematical models in controlling the spread of malaria. PhD thesis, University of Arizona, Tucson, Arizona, USA;.
28. N. Chitnis, J. M. Cushing, J. M. Hyman, (2006). Bifurcation analysis of a mathematical model for malaria transmission. SIAM J. Appl. Math. 67:24-45.
29. A. B. Okrinya, and J. L. Consul, (2019). Logistic mathematical model of Ebola virus disease with convalescence. International Journal of Applied Scientific and Research. 2(6):1-14.
30. J. S. Allen, (2007). An introduction to mathematical biology. Pearson Education Inc., Prentice Hall. Upper Saddle River, NJ07458.
31. M. O. Onuorah, and N. I. Akinwande, (2016). Sensitivity analysis of Lassa fever model. European Centre for Research Training and Development UK (www.eajournals.org).14(1)
32. and its Role on Global Stability. Biometrics, Statistics and Theoretical and Applied Mechanics Departments, Cornell University, Ithaca, NY 14853, USA.
33. S. Busenberg, P. Van den Driessche. Analysis of disease transmission model in a population with varying size, J. Maths.Biol. 28 (1990) 257-270.
34. M. Y. Li, H. I. Smith, L. Wang, Global stability of an SEIR epidemic model with vertical transmission, SIAMm. Appl. Math. 62 (1) (2001) 69-80.
35. S. M. Moghadas, A. B. Gumel, Global stability of a two-stage epidemic model with generalized non-linear incidence. Elsevier, Mathematics and Computers in Simulation 60 (2002) 107-118.
36. A. B. Okrinya, Mathematical modelling of Malaria Transmission and Pathogenesis, PhD Thesis, Loughborough University, 2015.