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Mathematical Modelling of Transmission Dynamics of Anthrax in Human and Animal Population.

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Abstract

Anthrax is an infectious disease that can be categorised under zoonotic diseases. It is caused by the bacteria known as Bacillus anthraces. Anthrax is one of the most leading causes of deaths in domestic and wild animals. In this paper, we develop and investigated a mathematical model for the transmission dynamics of the disease. Ordinary differential equations were formulated from the mathematical model. We performed the quantitative and qualitative analysis of the model to explain the transmission dynamics of the anthrax disease. We analysed and determined the model's steady states solutions. The disease-free equilibrium of the anthrax model is analysed for locally asymptotic stability and the associated epidemic basic reproduction number. The model's disease free equilibrium has shown to be locally asymptotically stable when the basic reproductive number is less than unity. The model is found to exhibit the existence of multiple endemic equilibria. Sensitivity analysis was performed on the model's parameters to investigate the most sensitive parameters in the dynamics of the diseases.

Keywords: Anthrax model, Basic reproductive number, Asymptotic stability, Endemic equilibrium, Sensitivity analysis.

1 Introduction

Bacillus anthraces is the bacteria responsible for anthrax disease. The disease is found naturally in soil and mostly affects wild and domestic and animals worldwide. Susceptible individuals can easily get sick with anthrax if they interact with infected animals or consumed contaminated dairy foods and animal products [27]. They are useful in the production of cheese, chemicals, yogurt and medicines. Bacteria play important roles as they manufacture and synthesize food particles in the digestive system to produce energy. However, bacteria organisms are responsible for many zoonotic diseases [29].

In recent times, mathematical models describing the phenomenon and dynamics of infectious diseases have played a key role in the control of diseases in epidemiology. Some of the models are able to explain the dynamics and mode of disease transmission [24, 21]. Many authors have proposed several nonlinear incidence rates to model the disease transmission dynamics. Complex transmission dynamics of some diseases such as periodic orbits, Hoff

bifurcations and multiple equilibrium have been described, they clearly explain and give a comprehensive qualitative illustration of the disease dynamics and give better analysis and implications for the control or prediction of diseases [5, 13].

Authors in [25, 2] formulated epidemic models on anthrax transmission in animals. They do not consider anthrax as a zoonotic disease. Anthrax affects both human and animal population in an environment. We further improve their work by developing an anthrax model that incorporate both human and animal populations. We incorporate vaccination as a compartment in the animal population. Authors in [25] analysed the compartmental model using the basic reproduction number but did consider sensitivity analysis. We improved the model by considering sensitivity analysis to see the contribution of each parameter on the model.

Authors in [27] investigated zoonotic diseases in Bangladesh. The findings revealed that, there were approximately 1415 human pathogens of which sixty-one percent are zoonotic related diseases. Authors in [15] investigated the effectiveness of constant and pulse vaccination policies using SIR model. From the theoretical results of their study under constant vaccination, the dynamics of the disease model is similar to dynamics without vaccination. Several studies have used the methods of optimal control theory in the formulation of the models [12][13]. However, a number of these studies focused on the effect of vaccination on the spread and transmission of the diseases as in the case of the authors in [18]. Also, authors in [3] studied a disease transmission model by considering the impact of a protective vaccine and came out with the optimal vaccine coverage threshold required for disease control and eradication. Moreover, in [9], optimal control was used to study a nonlinear SIR epidemic model with a vaccination strategy. Several mathematical modeling techniques have been employed to study the role of optimal control using SIR epidemic model [17, 30, 31]. [32], formulated an SIR epidemic model by considering vaccination as a control.

[16], also considered and applied optimal control to investigate the impact of chemo-therapy on malaria disease with infection immigrants and [4] applied optimal control methods associated with preventing exogenous reinfection based on a exogenous reinfection tuberculosis model. [11], considered and studied essential role of three basic types of control: personal protection, treatment, and mosquito reduction approaches in the control of malaria. Also, the authors developed a more general mathematical model in [23] of a vector-borne disease comprising of two vertebrate host species and one insect vector species.

[13] formulated an optimal control problem for an SIR epidemic model with saturated incidence and saturated treatment. Treatment and vaccination are basically the two main efforts that are considered to reduce the disease transmission. The concept of basic reproduction number was used to discuss the impacts of vaccination and treatment on the disease transmission. A compartmental and simulation models for evaluating Med-kits propositioning strategies for anthrax attack response was developed [6]. [6] developed a discrete-time compartmental difference equation model that analysed the policy. Their findings showed that distributing any number of Med-kits has a significant impact on the reduction of deaths expected.

Pathogenic bacteria and viruses can get access to waste matter and those designed for composting are not exception [7]. Authors in [21] formulated a model for the transmission dynamics of Listeriosis as a zoonotic disease in both animal and human populations. Zoonotic disease is an integral part both of human and animal population and therefore, the need to find alternative ways of combating these diseases.

Studies on experimental anthrax by Robert Koch in the early 1870s, demonstrated for the first time the bacterial origin of a specific disease and realised the spore stage that allows persistence of listeria monocytogenes in the environment [22]. Shortly afterwards, successful immunization of livestock against anthrax soon followed in 1880 by William Green field's. Even although Louis Pasteur's 1881 trial of a heat-cured anthrax vaccine in sheep would always be remembered as the initial use of a live vaccine [22]. The incidence of anthrax has actually increased in

African continent in recent years. This prompted the World Health Organization (WHO) to look for alternative measures of improving surveillance and control efforts [22]. The spread of diseases causes deaths of millions of people and cost of treatment of these diseases are of a great concern to every human endeavour. Public health is a major concern to the world at large. Adequate attention must be given to eradicate the spread of these diseases [14, 13]. Many studies in the literature have been carried out to determine the role of treatment and vaccination on the spread of diseases. A discrete-time epidemic model with vaccination for measles is formulated in [1]. Moreover, the effects of vaccination on the spread and transmission of periodic diseases was investigated using discrete-time model.

2 Anthrax Model Description and Formulation

The model divides the total human and vector populations at any time (t) into seven sub-populations (compartments) with respect to their disease status in the system.

The total vector population, represented by (N_v), is divided into sub-populations of Susceptible vector (S_v), Infectious vector (I_v), Vaccinated vector (V_v), and Recovered vector(R_v). The total vector population becomes:

$$N_v(t) = S_v(t) + V_v(t) + I_v(t) + R_v(t) \qquad (1)$$

The total human population also represented by N_h, is divided into sub-populations of Susceptible humans (S_h), Infected humans (I_h), and Recovered humans (R_h). The total human population is given by:

$$N_h(t) = S_h(t) + I_h(t) + R_h(t) \qquad (2)$$

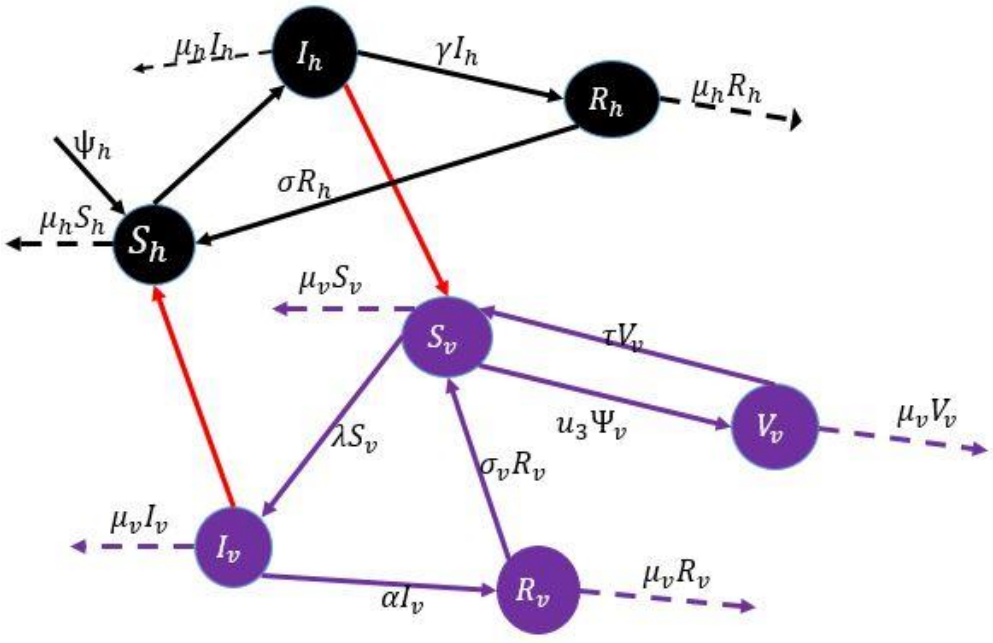

Figure 1 Flow chart for the anthrax disease transmission. The blue balls indicate the vector compartments and the black balls indicates the human compartments.

Susceptible animals include those that are at risk of getting the infection. Vaccinated animals include animals that are vaccinated before the Anthrax disease outbreak. Infectious animal compartment consists all animals that are showing the symptoms of the Anthrax disease. The recovered animal compartment consists of those that have recovered from the Anthrax disease and got temporal immunity. Susceptible human compartment includes individuals who are at risk of developing the infection. Infectious human compartment consists of individuals that are showing the symptoms of the Anthrax disease. The recovered human compartment comprises of individuals who have recovered from the Anthrax disease and got temporal immunity.

The Susceptible humans are recruited into the population at a rate ψ_h. Susceptible humans acquire Anthrax through inhalation of spores, ingestion of contaminated foods from infected animals, contact with infectious animals and humans at a rate $(I_h + I_v)\beta$. Individuals recover from the disease at a rate Υ. Humans who are infected with Anthrax die at a rate $\delta_h$and the recovered humans may lose immunity and return to the susceptible compartment at a rate σ_h. The natural death rate of the entire human compartments is μ_h.

The susceptible vector S_v are recruited into the population at a rate ψ_v. Anthrax can be acquired through contacts with infectious animals and humans at a rate $(I_h + I_v)\lambda$. The natural death rate of the animals is μ_v and the death rate as a result of the disease is δ_v. The animals recover at a rate α and a fraction of the vaccinated animals may move to the infected animal compartment at a rate $b\beta_m^*\lambda$ due to waning effect. Where $(1-b) \in [0,1]$ is the efficacy of the vaccine. This is because the animals may lose immunity and move back to the susceptible compartment at a rate τ.

Where $\beta_m^* = I_h + I_v$.

The following system of ordinary differential equations are obtained from the model flow diagram:

$$\begin{aligned}
\frac{dS_h}{dt} &= \psi_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h \\
\frac{dI_h}{dt} &= \beta_m^* \beta S_h - \gamma I_h - (\mu_h + S_h) I_h \\
\frac{dR_h}{dt} &= \gamma I_h - (\sigma_h + \mu_h) R_h \\
\frac{dS_v}{dt} &= (1-u_3)\psi_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v + \tau V_v \\
\frac{dI_v}{dt} &= \beta_m^* \lambda S_v + b\beta_m^* \lambda V_v - \alpha I_v - (\mu_v + \delta_v) I_v \\
\frac{dR_v}{dt} &= \alpha I_v - (\sigma_v + \mu_v) R_v \\
\frac{dV_v}{dt} &= u_3 \psi_v - (\tau - \mu_v) V_v - b\beta_m^* V_v
\end{aligned} \qquad (3)$$

3 Mathematical Analysis of the Anthrax model.

3.1 Positivity and Boundedness of Solutions

When dealing with human population model, we are aiming at getting non-negative solutions. Therefore, the conditions under which the system of differential equations under study has non-negative solutions is of great importance. The Anthrax model would be epidemically meaningful on condition that all the solutions with non-negative initial data remain non-negative at every time. The concept of the derivative of a function would be applied. The derivative of a function at a point is one of the basic properties that determines the behaviour of that particular function even when that function is unknown. If the derivative of a function at a point is positive, then the function is said to be increasing at that point. If the derivative of the function at a point is negative, then it is said to be decreasing and if the derivative of the function at a point is equal to zero, then the function is constant.

Let

$\Pi = \{(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), R_v(t), V_v(t))\} \in \mathbb{R}_+^7$:

$(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), R_v(0), V_v(0)) > 0$, then the solution of

$\{(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), R_v(t), V_v(t))\}$ are non-negative for all time $t \geq 0$.

If $S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), R_v(0), V_v(0)$ are non-negative, then

$S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), R_v(t), V_v(t)$ are also non-negative for all time $t > 0$.

Considering the human population in the model:

The total human population at any time (t) is given by:

$$N_h(t) = S_h(t) + I_h(t) + R_h(t)$$

This is given by;

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \qquad (4)$$

$$\frac{dN_h}{dt} = \psi_h - \mu_h S_h - \beta^* \beta S_h + \sigma_h R_h + \beta_m^* \beta S_h - \Upsilon I_h - (\mu_h + \delta_h) I_h + \Upsilon I_h - (\mu_h + \sigma_h) I_h$$

The above can be written as;

$$\frac{dN_h}{dt} = \psi_h - \mu_h S_h + \sigma_h R_h - (\mu_h + \delta_h) I_h - (\sigma_h + \mu_h) R_h$$

In the absence of mortality due to Anthrax infections, the above equation becomes;

$$\frac{dN_h}{dt} \leq \psi_h - \mu_h N_h \qquad (5)$$

Solving the ordinary differential equation;

$$\psi_h = \mu_h N_h \geq A e^{-\mu_h t}$$

where A is constant. Applying the initial condition,

$$N_h(0) = N_h(0)$$

We obtain the relation;

$$\psi_h - \mu_h N_h(0) = A$$

Therefore;

$$\psi_h - \mu_h N_h \geq (\psi_h - \mu_h N_h(0)) e^{-\mu_h t}$$

$$N_h \leq \frac{\psi_h}{\mu_h} - \left(\frac{\psi_h - \mu_h N_h(0)}{\mu_h}\right) e^{-\mu_h t}$$

$t \to \infty$ the population size $N_h \to \frac{\psi_h}{\mu_h}$

This implies that; $0 \leq N_h \leq \frac{\psi_h}{\mu_h}$ and $N_h(t) \leq \frac{\psi_h}{\mu_h}$

Also, if $N_h(0) \leq \frac{\psi_h}{\mu_h}$, then $N_h(t) \leq \frac{\psi_h}{\mu_h}$

$$\text{Therefore,} \quad \Pi_h = \left\{(S_h, I_h, R_h) \in \mathbb{R}_3^2 : S_h + I_h + R_h \leq \frac{\psi_h}{\mu_h}\right\} \qquad (6)$$

The total vector(livestock) population at any time (t)is given by:

$N_v(t) = S_v(t) + V_v(t) + I_v(t) + R_v(t)$

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dI_v}{dt} + \frac{dR_v}{dt} + \frac{dR_v}{dt} \qquad (7)$$

In the absence of mortality due to Anthrax infections, the above equation becomes;

$\frac{dN_v}{dt} \leq \psi_v - \mu_v N_v$

Solving the ordinary differential equation;

$\psi_v = \mu_v N_v \geq Ae^{-\mu_v t}$

where A is constant. Applying the initial condition,

$N_v(0) = N_v(0)$

We obtain the relation;

$\psi_v - \mu_v N_v(0) = A$

Therefore;

$\psi_v - \mu N_v \geq (\psi_v - \mu_v N_v(0))e^{-\mu_v t}$

$N_v \leq \frac{\psi_v}{\mu_v} - (\frac{\psi_v - \mu_v N_v(0)}{\mu_v})e^{-\mu_v t}$

$t \to \infty$ the population size $N_v \to \frac{\psi_v}{\mu_v}$

This implies that; $0 \leq N_v \leq \frac{\psi_v}{\mu_v}$ and $N_v(t) \leq \frac{\psi_v}{\mu_v}$

Also, if $N_v(0) \leq \frac{\psi_v}{\mu_v}$, then $N_v(t) \leq \frac{\psi_v}{\mu_v}$

Therefore, $\Pi_v = \left\{(S_v, I_v, R_v) \in \mathbb{R}_3^3 : S_v + I_v + R_v \leq \frac{\psi_v}{\mu_v}\right\}$ (8)

$$\Pi = \Pi_h \times \Pi_v \subset \mathbb{R}_+^3 \times \mathbb{R}_4^4 \quad (9)$$

$$\Pi_h = \left\{(S_h, I_h, R_h) \in \mathbb{R}_3^3 : S_h + I_h + R_h \leq \frac{\psi_h}{\mu_h}\right\} \quad (10)$$

$$\Pi_v = \left\{(S_v, I_v, R_v, V_v) \in \mathbb{R}_+^4 : S_v + I_v + R_v + V_v \leq \frac{\psi_v}{\mu_h}\right\} \quad (11)$$

3.2 Disease-free equilibrium for the Anthrax model.

The disease-free equilibrium of the system of ordinary differential equations in (2.1) only exists when $u_1 = 0$ and all other controls are held constant.

This is computed by setting the system of differential equations in (2.1) to zero. This is given by:

$$\xi_o = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*, R_v^*, V_v^*) \quad (12)$$

At disease free equilibrium (DFE), there are no infections and recovery.

$$\left.\begin{matrix} I_h^* = 0 \\ R_h^* = 0 \end{matrix}\right\} \left.\begin{matrix} I_v^* = 0 \\ R_v^* = 0 \end{matrix}\right\}$$

$$\frac{dS_h}{dt} = \psi_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h = 0 \quad (13)$$

$$S_h^* = \frac{\psi_h}{\mu_h} \quad (14)$$

Now considering the vector (Livestock) population: At disease free equilibrium, there are no infections and recovery.

$$\frac{dV_v}{dt} = u_3 \psi_v - (\tau + \mu_v) v_v - b\lambda \beta_m^* V_v = 0$$

$$u_3 \psi_v - (\tau + \mu_v) V_v = 0$$

$$V_v^* = \frac{u_3 \psi_v}{\tau + \mu_v}$$

Also, from the relation;

$$\frac{dS_v}{dt} = (1 - u_3)\psi_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v + \tau V_v = 0$$

$$(1-u_3)\psi_v - \mu_v S_v + \tau V_v = 0$$

$$S_v^* = \frac{(1-u_3)\psi_v + \tau V_v}{\mu_v}$$

$$S_v^* = \frac{\psi_v(\tau + \mu_v(1-u_3))}{\mu_v(\tau+\mu_v)}$$

$$\xi_o = \left(\frac{\psi_h}{\mu_h}, 0, 0, \frac{\psi_v(\tau+\mu_v(1-u_3))}{\mu_v(\tau+\mu_v)}, 0, 0, \frac{u_3\psi_v}{\tau+\mu_v}\right) \qquad (15)$$

3.3 The Basic Reproductive Number

Using the Next Generation Matrix, the linear stability of the disease-free equilibrium (ξ_0) can be established. This is done by calculating the basic reproductive number using the next generation matrix. The basic reproductive number or rate is the number of secondary cases produced on average by one infected animal or person when all are susceptible. It combines the biology of infections with the social and behaviour of the factors influencing contact rate. The basic reproduction rate gives the number of secondary cases one infectious individual will produce in a population consisting only of susceptible individuals [28, 20]. The basic reproductive number is the threshold parameter that governs the spread of a disease.

The next-generation matrix is defined as; $K = FV^{-1}$ and $R_0 = \rho(FV^{-1})$. Where $\rho(FV^{-1})$ denotes the spectral radius of $\rho(FV^{-1})$.

The basic reproductive number R_0, is defined as the spectral radius of the next-generation matrix.
The spectral radius of a matrix A is defined as the maximum of the absolute values of the eigenvalues of the matrix $A: \rho(A) = \sup\{|\lambda|\} : \lambda \in \rho(A)$ where $\rho(A)$ represents the set of eigenvalues of the matrix A.

Using the Next Generation Matrix, we consider only the infective classes in the system of differential equations in (3):

$$\frac{dI_h}{dt} = (I_h + I_v)\beta S_h - \gamma I_h - (\mu_h + \delta_h)I_h \qquad (16)$$

$$\frac{dI_v}{dt} = (I_h + I_v)\lambda S_v + (I_h + I_v)b\lambda V_v - \alpha I_v - (\mu_v + \delta_v)I_v \qquad (17)$$

Let f=$\begin{bmatrix} (I_h + I_v)\beta S_h \\ (I_h + I_v)\lambda S_v + (I_h + I_v)b\lambda V_v \end{bmatrix}$

and $v = \begin{bmatrix} \gamma I_h + (\mu_h + \delta_h)I_h \\ \alpha I_v + (\mu_v + \delta_v)I_v \end{bmatrix}$

Let F and V be represented by;

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \beta S_h & \beta S_h \\ \lambda S_v + b\lambda V_v & \lambda S_v + b\lambda V_v \end{bmatrix} \quad (18)$$

$$V = \begin{bmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \gamma + (\mu_h + \delta_n) & 0 \\ 0 & \alpha + (\mu_v + \delta_v) \end{bmatrix} \quad (19)$$

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \beta S_h^* & \beta S_h^* \\ \lambda S_v^* + b\lambda V_v^* & \lambda S_v^* + b\lambda V_v^* \end{bmatrix} \quad (20)$$

$$V = \begin{bmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \gamma + (\mu_h + \delta_h) & 0 \\ 0 & \alpha + (\mu_v + \delta_v) \end{bmatrix} \quad (21)$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma + (\mu_h + \delta_n)} & 0 \\ 0 & \frac{1}{\alpha + (\mu_v + \delta_v)} \end{bmatrix} \quad (22)$$

By computing the product of FV^{-1};

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_h^*}{\gamma + (\mu_h + \delta_n)} & \frac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} \\ \frac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} & \frac{\lambda S_v^* + b\lambda V_v^*}{\alpha + (\mu_v + \delta_v)} \end{bmatrix} \quad (23)$$

Finding the eigenvalues of the matrix;

$$\begin{vmatrix} \frac{\beta S_h^*}{\gamma + (\mu_h + \delta_n)} - A & \frac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} \\ \frac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} & \frac{\lambda S_v^* + b\lambda V_v^*}{\alpha + (\mu_v + \delta_v)} - A \end{vmatrix} = 0 \quad (24)$$

$$A^2 - \left[\left(\frac{\lambda S_v^* + b\lambda V_v^*}{\alpha + (\mu_v + \delta_v)}\right) + \left(\frac{\beta S_h^*}{\gamma + (\mu_h + \delta_n)}\right)\right] A = 0 \quad (25)$$

$A_1 = 0$ and

$$A_2 = \left[\left(\frac{\lambda S_v^* + b\lambda V_v^*}{\alpha + (\mu_v + \delta_v)}\right) + \left(\frac{\beta S_h^*}{\gamma + (\mu_h + \delta_n)}\right)\right]$$

The dominant eigenvalue is A_2, therefore

$$R_{hv} = \left[\left(\frac{\beta S_h^*}{\gamma + (\mu_h + \delta_n)}\right) + \left(\frac{\lambda S_v^* + b\lambda V_v^*}{[\alpha + (\mu_v + \delta_v)]}\right)\right] \qquad (26)$$

But $S_h^* = \frac{\psi_h}{\mu_h}$, $S_v^* = \frac{\psi_h(\tau+\mu_v(1-u_3))}{\mu_v(\tau+\mu_v)}$ and $V_v^* = \frac{u_3\psi_v}{\tau+\mu_v}$

$$R_{hv} = \left(\frac{\beta\psi_h}{\mu_h[\gamma + (\mu_n + \delta_n)]}\right) + \left(b\lambda\left(\frac{\lambda_v}{\mu_v} - \frac{2\psi_v u_3}{\tau + \mu_v}\right)\right) \qquad (27)$$

Where R_{hq} and R_{vq} are the reproductive numbers for human and animal respectively.

$$R_{hq} = \left(\frac{\beta\psi_h}{\mu_h[\gamma + (\mu_h + \delta_h)]}\right) \qquad (28)$$

$$R_{vq} = \left(b\lambda\left(\frac{\psi_v}{\mu_v} - \frac{2\psi_v u_3}{\tau + \mu_v}\right)\right) \qquad (29)$$

3.4 Global stability of the disease-free equilibrium

Theorem: If $R_{hv} < 1$, the disease-free equilibrium is globally asymptotically stable in the interior of Ω.

Proof: Considering the Lyapunov function below;

$$P(t) = (\alpha + \mu_v + S_v)I_h + (\gamma + \mu_h + \delta_h)I_v \qquad (30)$$

By computing the time derivative of P along the solutions of the system of ordinary differential equations in (3), the following is obtained,

$$\frac{dP(t)}{dt} = (\alpha + \mu_v + \delta_v)\frac{dI_h}{dt} + (\gamma + \mu_h + \delta_h)\frac{dI_v}{dt} \qquad (31)$$

$$= (\beta S_h(I_h + I_v) - \gamma I_h - (\mu_h + \delta_h)I_h) + \lambda S_v(I_h + I_v) + b(I_h + I_v)\lambda V_v - \alpha I_v - (\mu_v + \delta_v)I_v]$$

$$\leq (\alpha + \mu_v + \delta_v)\frac{\beta\psi_h I_h}{\mu_h} + (\alpha + \mu_v + \delta_v)\frac{\beta\psi_h I_v}{\mu_h} - (\alpha + \mu_v + \delta_v)(\gamma + \mu_h + \delta_h)I_h$$

$$+ I_h(\gamma + \mu_v + \delta_v)\left(\frac{\lambda\psi_v(\tau + \mu_v(I - u_3))}{\mu_v(\tau + u_v)}\right) + I_v(\gamma + \mu_h + \delta)\left(\frac{\lambda\psi_v(\tau + \mu_v(I - u_3))}{\mu_v(\tau + u_v)}\right)$$

$$+ I_h(\gamma + \mu_h + \delta_h)\left(\frac{bu_3\lambda\psi_v}{\tau + \mu_v}\right) + I_v(\gamma + \mu_h + \delta)\left(\frac{bu_3\lambda\psi_v}{\tau + \mu_v}\right) - I_v(\gamma + \mu_h + \delta)(\alpha + \mu_v$$

$$+ \delta_v)$$

$$\frac{dP(t)}{dt} \leq -I_h(\gamma + \mu_h + \delta_h)(\alpha + \mu_v + \delta_v)(1 - R_{hv}) - I_v(\gamma + \mu_h + \delta_h)(\alpha + \mu_v + \delta_v)(1 - R_{hv}$$

$$= -(I_h + I_v)(\gamma + \mu_h + \delta_h)(\alpha + \mu_v + \delta_v)(1 - R_{hv}) \qquad (32)$$

The time derivative of P along the solutions of the system of differential equations in (3) gives the following:

$\frac{dP(t)}{dt} \leq 0$, if and only if $R_{hv} < 0$.

$\frac{dP(t)}{dt} = 1$ if and only if $I_h + I_v = 0$ or $R_{hv} = 1$

Therefore, the highest compact invariant set in $S_h, I_h, I_v \in \Omega$, $\frac{dP(t)}{dt} = 0$ if $R_{hv} \leq 1$.

is the singleton ξ_0.

This implies that ξ_0 is globally asymptotically stable in Ω. By LaSalle's invariant principle[10].

3.6 Global stability of endemic equilibrium

The Global behaviour of the system of differential equations in equation (3) is analysed.

The system of differential equations in equation (3), is said to have a unique endemic equilibrium if $R_{hv} \leq 1$, and it is globally asymptotically stable.

The endemic equilibrium can only exists if and only if $R_{hv} \leq 1$. So by letting $R_{hv} \leq 1$, it implies that the endemic equilibrium exists.

Considering the non-linear Lyapunov function bellow;

$$L = S_h^{**}\left(\frac{S_h}{S_h^{**}} - ln\frac{S_h}{S_h^{**}}\right) + I_h^{**}\left(\frac{I_h}{I_h^{**}} - ln\frac{I_h}{I_h^{**}}\right) + \frac{g_1 R_h^{**}}{\gamma}\left(\frac{R_h}{R_h^{**}} - ln\frac{R_h}{R_h^{**}}\right) + S_v^{**}\left(\frac{S_v}{S_v^{**}} - ln\frac{S_v}{S_v^{**}}\right)$$
$$+ I_v^{**}\left(\frac{I_v}{I^{**}} - ln\frac{I_v}{I_v^{**}}\right) + R_v^{**}\left(\frac{R_v}{R_h^{**}} - ln\frac{R_v}{R^{**}}\right) + V_v^{**}\left(\frac{V_v}{V_v^{**}} - ln\frac{V_v}{V_v^{**}}\right) \qquad (33)$$

Where

$g_1 = \gamma + (\mu_h + \delta_h)$

$g_2 = (\sigma_2 + \mu_h)$

$g_3 = \alpha + (\mu_v + \delta_v)$

$g_4 = (\sigma_v + \mu_v)$

$$\frac{dL}{dt} = \left(1 - \frac{S_h^{**}}{S_h}\right)\frac{dS_h}{dt} + \left(1 - \frac{I_h^{**}}{I_h}\right)\frac{dI_h}{dt} + \frac{g_1}{\gamma}\left(1 - \frac{R_h^{**}}{R_h}\right)\frac{dR_h}{dt} + \left(1 - \frac{S_v^{**}}{S_v}\right)\frac{dS_v}{dt} + \left(1 - \frac{I_v^{**}}{I_v}\right)\frac{dI_v}{dt} + \left(1 - \frac{R_v^{**}}{R_v}\right)\frac{dR_v}{dt} +$$
$$\left(1 - \frac{V_v^{**}}{V_v}\right)\frac{dV_v}{dt} \qquad (34)$$

$$\frac{dL}{dt} = \left(1 - \frac{S_h^{**}}{S_h}\right)[\psi_h + \sigma_h R_h^{**} + \beta\beta_m^{**} S_h^{**} + \mu_h S_h^{**} - \psi_h - \sigma R_h - \beta\beta_m S_h - \mu_h S_h] + \left(1 + \frac{I_h^{**}}{I_h}\right)[\beta\beta_m S_h -$$

$$g_1 I_h] + \frac{g_1}{\gamma}\left(1 - \frac{R_h^{**}}{R_h}\right)[\gamma I_h - g_2 R_h] + \left(1 - \frac{S_v^{**}}{S_v}\right)[(1-u_3)\psi_v + \lambda\beta\beta_m^{**} S_v^{**} + \mu_v S_v^{**} + \sigma_v R_v^{**} + \tau V_v^{**} -$$

$$(1-u_3)\Lambda_v - \lambda\beta_m S_v - \mu_v S_v - \sigma_v R_v - \tau V_v] + \left(1 - \frac{I_v^{**}}{I_v}\right)[\lambda\beta_m S_v + b\lambda\beta_m V_v - g_3 I_v] + \frac{g_3}{\alpha}\left(1 - \frac{R_v^{**}}{R_v}\right)[\alpha I_v -$$

$$g_4 R_v] + \left(1 - \frac{V_v^{**}}{V_v}\right)[u_3\Lambda_v + b\lambda\beta_m^{**} V_v^{**} + (\tau + \mu_v)V_v^{**} - u_3\psi_v - b\lambda\beta_m V_v + (\tau + \mu_v)V_v]$$

Basically, however, the arithmetic mean value exceeds the geometric mean value[26]. This follows that;

$$2 - \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}} \leq 0$$

$$1 - \frac{R_h}{R_h^{**}} \leq 0$$

$$1 - \frac{R_h}{R_h^{**}} + \frac{g_1 g_2 S_h}{\gamma S_h^{**}}\left(1 - \frac{R_h^{**}}{R_h}\right) \leq 0$$

$$1 - \frac{\beta_m}{\beta_h^{**}} - \frac{S_h^{**}}{S_h} - \frac{S_h \beta_m I_h^{**}}{S_h^{**}\beta_m^{**}} \leq 0$$

$$1 - \frac{I_h}{I_h^{**}} - \frac{\gamma I_h}{I_h^{**}}\left(1 - \frac{R_h^{**}}{R_h}\right) \leq 0$$

$$2 - \frac{S_v^{**}}{S_v} - \frac{S_v}{S_v^{**}} \leq 0$$

$$1 - \frac{S_v^{**}}{S_v} - \frac{R_v}{R_v^{**}} + \frac{R_v S_v^{**}}{R_v^{**} S_v} \leq 0$$

$$1 - \frac{S_v^{**}}{S_v} - \frac{V_v}{V_v^{**}} + \frac{V_v S_v^{**}}{V_v^{**} S_v} \leq 0$$

$$1 - \frac{S_v^{**}}{S_v} + \frac{\beta_m}{\beta_h^{**}} - \frac{S_v \beta_m I_v^{**}}{S_v^{**}\beta_m^{**} I_v} \leq 0$$

$$1 + \frac{I_v}{I_v^{**}} - g_3\alpha\frac{I_v}{I_v^{**}} - g_3\alpha\frac{I_v R_v^{**}}{I_v^{**} R_v} \leq 0$$

$$1 - \frac{R_v}{R_v^{**}} \leq 0$$

$$2 - \frac{V_v^{**}}{V_v} - \frac{V_v}{V_v^{**}} \leq 0$$

$$1 - \frac{V_v^{**}}{V_v} + \frac{\beta_m}{\beta_h^{**}} - \frac{V_v \beta_m I_v^{**}}{V_v^{**} \beta_h^{**} I_v}$$

From the assumption that all the model parameters are non-negative, it implies that the derivative of the Lyapunov function is less than zero , $\frac{dL}{dt} \leq 0$, if the basic reproduction number of the system of differential equation in equation (2.1) is greater than one $R_{hv} > 1$. Therefore by LaSalle's Invariant Principle[10, 20], as t approaches infinity, all the solution of the equations of the system of differential equations in the model approaches the endemic equilibrium point if $R_{hv} > 1$.

4 Sensitivity analysis of the Anthrax model

Basically, the essence of sensitivity analysis is to determine how robust a model is to parameter values. This is usually done to help identify the parameters with high impact on the basic reproduction number (R_{hv}). The basic reproduction number is usually analysed to find out whether or not treatment of the infective, mortality and vaccination could help in the control or eradication of the disease in the population[20]

The normalised forward sensitivity index of a variable, q, which depends differentially on a parameter, r, defined as:

$$r_x^y = \frac{\partial y}{\partial x} \times \frac{x}{y} \qquad (35)$$

4.1 Sensitivity indices of the basic reproduction number.

In epidemiological models, the value of the basic reproductive number determines the ability of the disease or infection to spread within the population. We will determine the reduction in infection due to the diseases by computing the sensitivity indices of the basic reproduction Number R_{hv} with respect to the parameter values in the model. The sensitivity indices serve as determinants of the significance of each parameter in the dynamics and prevalence of the diseases. They measure the change in model variables when a parameter changes. In this study, we will compute the sensitivity indices of R_{hv} to parameter values for the model which will be estimated from data available or already published papers in the literature. Considering the thirteen different parameters of the system of differential equations in model (3), we therefore derive the sensitivity of $R_{hv}$to each of the parameters in the model. We incorporated sensitivity analysis in our model and an improvement of the work done by [25, 2]. This determine the sensitive parameters in the model which comprises of both human and animal populations.

The sensitivity indices of the basic reproduction number R_{hv} ,with respect to each of the parameters of the system of differential equations in model (3), are given in the table below:

Parameter	Description	Sensitivity Index(+ve/-ve)
α	animals recovery rate	-ve
β	Human transmission rate	+ve
λ	Aanimal transmission rate	+ve
ϒ	animals recovery rate	-ve
τ	Waning rate	+ve
b	Vaccine efficacy	+ve
u_3	Proportion vaccinated	-ve
ψ_h	Human recruitment	+ve
μ_h	Human natural death rate	-ve
δ_h	Disease induced death rate	-ve
ψ_v	Animal recruitment rate	+ve
μ_v	Animal natural death rate	-ve
δ_v	Disease induced death rate	-ve

Table 2 Sensitivity indices of parameters to R_{hv}.

The detailed sensitivity analysis of the basic reproductive number (R_{hv})as a result of evaluation to the other parameters of the model in Figure 2 shows that increasing μ_h, μ_v and αwould decrease the basic reproduction number R_{hv}. Moreover, decreasing μ_h μ_v and α would increase the basic reproductive number R_{hv}. Also, by increasing

ψ_h, ψ_v, β and λ would cause an increase in the basic reproduction number R_{hv} and by decreasing ψ_h, ψ_v, β and λ would cause a decrease in the basic reproduction number R_{hv}.

5 Numerical Results

In this section, numerical simulations are used to show the impact of vaccination using the fourth order Range-Kutta scheme on the state equations. [20, 8]. The state equations are solved over a simulated period using Range-Kutta fourth order scheme.

Parameter	Value	Reference
α	0.0025	assumed
β	0.0001	[19]
ϒ	0.75	[19]
λ	0.00005	assumed
τ	0.004	assumed
b	0.003	assumed
ψ_h	0.2	[19]
μ_h	0.0001	assumed
δ_h	0.2	Steven Kim (Health-line, Dec 2015)
ψ_v	0.005	[19]
μ_v	0.0004	[19]
δ_v	0.45	assumed

Table 3 Variable and parameter values of Anthrax model.

5.1 Existence of backward bifurcation.

In this section, we show the numerical simulation of model (3) by indicating the existence of backward bifurcation. Biologically, this means that the necessary condition for anthrax eradication when the basic reproduction number is less than one is no longer applicable. Backward bifurcation in model (3), means it is not sufficient to look at the dynamics of anthrax based on only reproduction number.

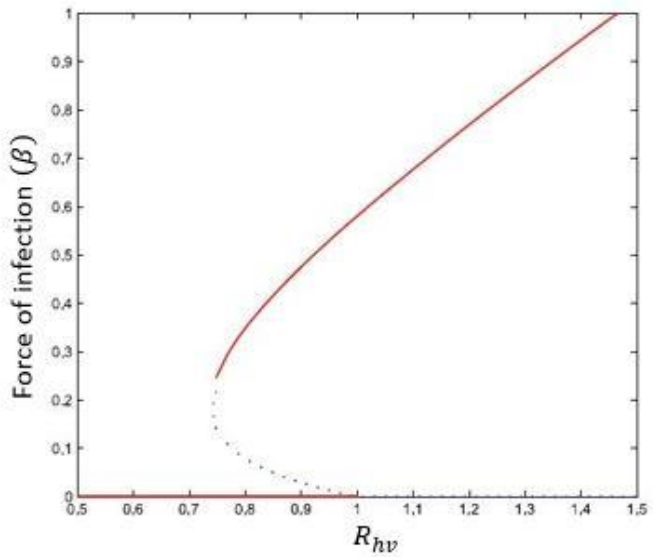

Figure 2 Simulation of model (3)indicating the existence of backward bifurcation of the endemic equilibrium. The red line indicates stable equilibrium and the blue dotted lines indicates an unstable equilibrium.

5.2 Simulations showing effects of force of infection (β) on infectious vector and human population.

In this section, we simulate the model in equation (3), to see the effects of the force of infection (β)on the infectious population of both the human and the vector. Figure 3 shows the effects of the force of infection on the population of the infectious vector. As the value of (β)decreases, the population of the infectious vector reduces. Figure 4 shows the effects of the force of infection on the infectious human population. A reduction in the value of (β), reduces the number of the infectious human population.

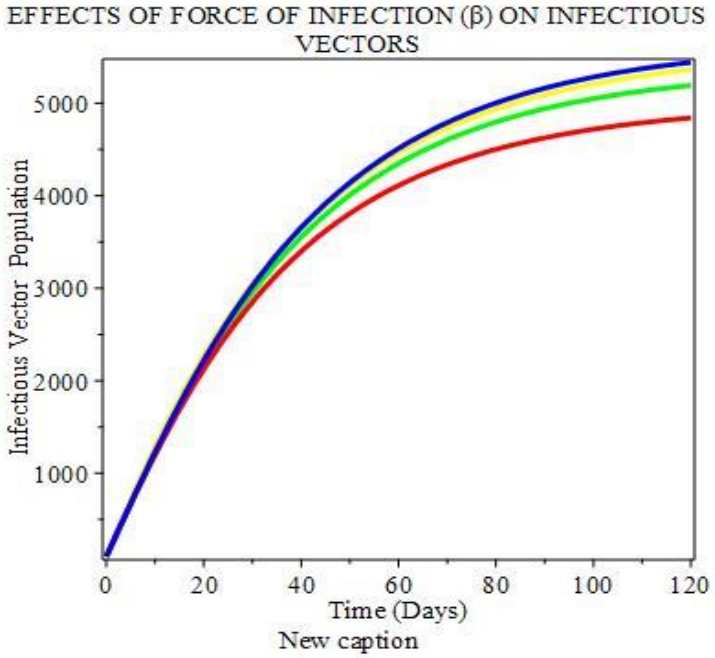

Figure 3 Simulations of the anthrax model indicating the effects of force of infections on infectious vectors.

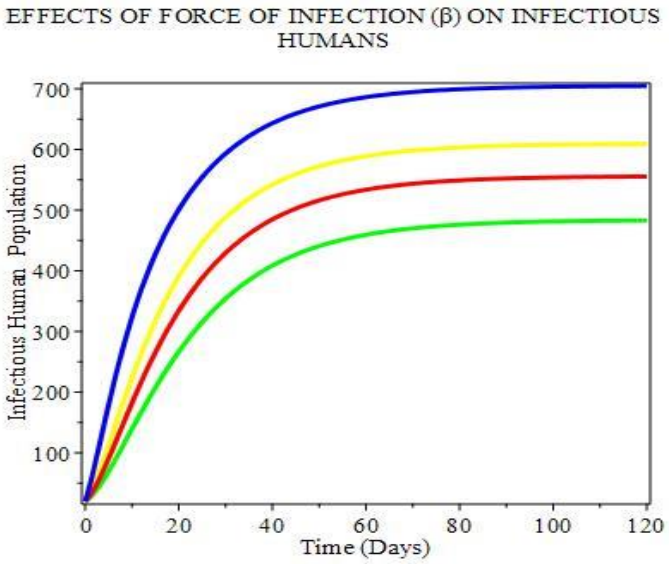

Figure 4 Simulations of anthrax model indicating the effects of force of infection on infectious humans.

5.3 Simulations of anthrax model showing infectious vector and infectious human populations.

The diagrams show the simulations of the anthrax model indicating the pattern of the infectious vector population and the infectious human populations. As the number of susceptible vector and human populations increases in the system, there are higher chances of individuals and animals to get infected with the disease since interactions in the

system increases. The model assumes the concept of mass action.

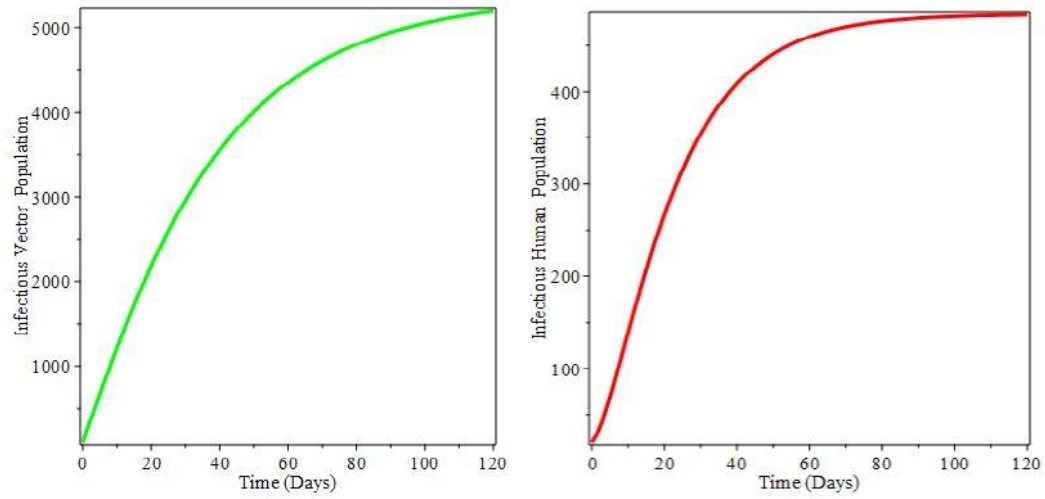

Figure 5 Simulation of Anthrax model showing Infectious vector and Infectious human populations.

5.4 Simulations of anthrax model showing susceptible vector and susceptible human populations

The number of susceptible vector and susceptible human populations increases in the system as the result of the model been an open system. There are more vector and humans coming into the population and this increases the number of susceptible vector and human populations.

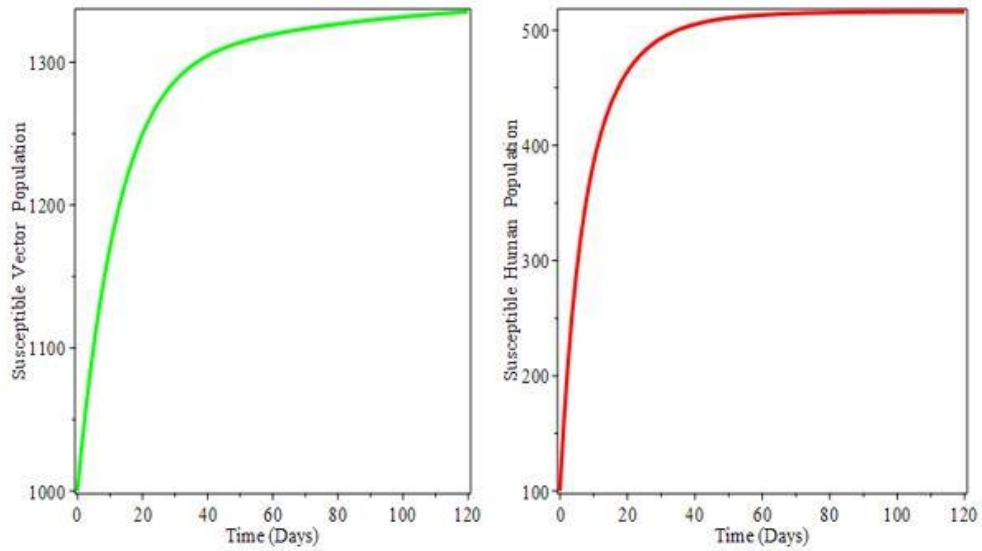

Figure 6 Simulation of Anthrax model showing Susceptible vector and Susceptible human populations.

5.5 Simulations of anthrax model showing recovered vector and recovered human population

The recovered human population decreases steadily and there is an equilibrium at some point in time. This can be as a result of the number of individuals recovering from the disease are equal to the number of people getting infected from the anthrax disease. In the vector population, the number of animals recovering from the disease decreases with time and a sharp increase in the number of recovery. This could be as a result of the vaccination of the susceptible vector populations.

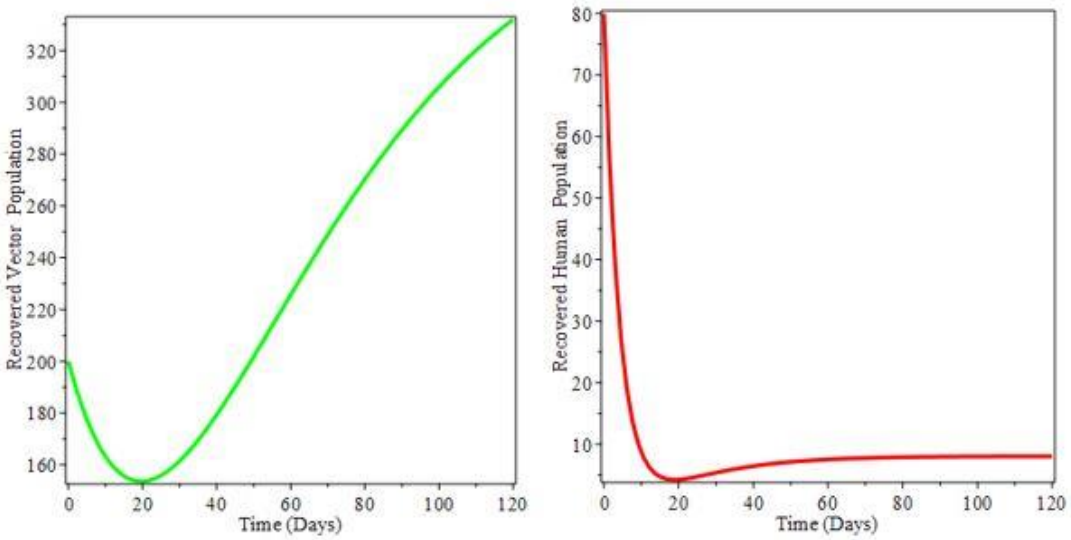

Figure 7 Simulation of Anthrax model showing the Recovered vector and Recovered human populations.

6 Results and Discussion

In this paper, we developed a compartmental model for the transmission dynamics of Anthrax infections by addition of vaccination of susceptible vector (Livestock) compartment. We investigate the impact of the vaccination compartment on the transmission dynamics of the disease. We derived the basic reproductive number and established that our model has a globally stable infection- free equilibrium when the basic reproductive number is less than one. The model exhibited an existence of multiple endemic equilibrium. The implication is that the disease can best be eradicated if the basic reproductive number is always less than one. The advantage of our model is the incorporation of both human and animal population in the model. The basic reproductive number was computed with respect to both the human and animal compartments.

We performed the sensitivity analysis of the basic reproductive number to each of the parameters to determine which parameter is more sensitive. This analysis was incorporated into our model to compare it to the work done by [25, 2]. This determine the sensitive parameters in the model which comprises of both human and animal populations. The work done by [25, 2] critical examines the anthrax disease in only animal population. Our model examines anthrax in both animal and human populations and the results showed that; by decreasing the animal recovery rate, it would cause an increase in the basic reproduction number. Moreover, by increasing the human recruitment rate, it would cause an increase in the basic reproduction number. Also, decreasing human recruitment rate, livestock recruitment rate, livestock transmission rate and human transmission rate would cause a decrease in the basic reproduction number. Increasing human recruitment rate, livestock recruitment rate, livestock transmission rate and human transmission rate would cause an increase in the basic reproduction number.

The advantage of our model is that it explains the dynamics of the transmission of the anthrax disease by showing that the parameters in both human and animal contributes to the disease transmission dynamics. However, the incorporation of the vaccinated compartment in our has helped improved the work of [25, 2] as the results of our sensitivity analysis shows the effects of the vaccinated compartment. The advantage of our model to the existing models is that, it compares the rate of infections and rate of recovery of anthrax disease in both the human and animal populations. From our numerical results, it showed that the rate of recovery of the disease in animals is faster than the rate of recovery of the disease in humans.

Data availability statement

Data supporting this model are from previously published articles and they have been duly cited in this paper.

Parameter values taken from published articles are cited at relevant places within the text as references.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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