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A Mathematical Model Analysis on Anthrax Disease Considering Environmental Spore Concentration

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Abstract: This study presents a mathematical model of an anthrax disease, which focuses on the role of the concentration of spores in the environment. The main objective is to analyse the dynamics of the disease and to assess the stability of its equilibrium points—in particular, those of the endemic and anthrax-free states. The relevance of this research is its potential to inform public health strategies for the management of anthrax attacks. This study used the Castillo–Chavez and LaSalle invariant principles to establish the boundedness, positivity, and existence of the solutions to the system. In addition, the basic reproduction number was calculated (R_0) using the next-generation matrix approach and performed sensitivity analyses to assess the effect of parameters such as host-to-host transmission coefficient (β_1), environmental transmission coefficient (β_2), and spore production rate (p) on the R_0 parameter. To confirm my analytical findings, numerical simulations have been performed to confirm that a decrease in these parameters leads to a decrease in reproductive rate. The simulation shows that if $R_0 < 1$ is stable both locally and globally, the anthrax disease-free equilibrium is stable, indicating the presence of effective control measures. On the other hand, the endemic equilibrium is stabilised at $R_0 > 1$, which highlights the need for immediate intervention.

Keywords: anthrax; environmental spore; sensitivity analysis and stability analysis

1. Introduction

Bacillus anthracis, a bacterium capable of producing spores that persist in the environment for extended periods, is the causative agent of anthrax, a severe and formidable infectious disease. Anthrax is a zoonotic disease that affects herbivores, including horses, cattle, and goats, with the potential for transmission to humans. The causative agent is the bacterium *Bacillus anthracis*, which is disseminated via animal hosts and insect vectors. Anthrax spores may enter the human body through the respiratory tract or cutaneous lesions, typically manifesting an incubation period ranging from one to several days [1]. The Gram-positive bacterium *Bacillus anthracis* is the causative agent of the global zoonotic disease anthrax. Transmission occurs primarily through environmental spores, which are capable of persisting for extended durations [2]. Anthrax spores can survive for up to two centuries due in part to their exceptional resilience to pH extremes, heat and cold, dehydration, and various chemical agents [3]. Furthermore, bovine anthrax infections may occasionally progress without the manifestation of clinical symptoms [4]. Under these circumstances, the prevention of anthrax outbreaks may be significantly contingent upon the duration of the incubation period preceding mortality.

This condition primarily impacts agricultural populations residing in economically disadvantaged tropical regions [5]. The presence of anthrax within a region poses significant challenges to the local community, as it jeopardizes both public health and economic stability, particularly for those whose livelihoods depend upon livestock [6]. Anthrax diminishes the efficiency with which inputs are converted into outputs, thereby exerting a substantial impact on the economy [7]. Anthrax represents a highly contagious zoonotic disease capable of causing significant mortality in animal populations, substantial economic losses, and widespread social disruption [8].

Mathematical models are crucial for understanding how anthrax spreads within a community. They also help us better grasp the dynamics of anthrax transmission. Numerous mathematical models have been put up to investigate how various elements affect the dynamics of anthrax spread. The authors in [4] constructed an enhanced version of

the anthrax transmission model of [9], taking into account a nonlinear ratio-dependent disease transmission function. The entire population is divided into four categories in the suggested model of [4]: susceptible, infectious, recovered, and vaccinated. Even if there is minimal human involvement in the animal-to-animal spread of anthrax disease, it is important to note that disease transmission among animals is the primary factor. And investigated a proper vaccination policy can effectively control anthrax transmission in the animal population.

The authors in [10] divided the model into groups in order to develop a mathematical model. The total animal population was stratified into sub-compartments consisting of susceptible individuals, those infected with anthrax, those infected with listeriosis, those co-infected with both pathogens, and individuals who had recovered from anthrax, listeriosis, or both. Similarly, the vector population was partitioned into distinct groups comprising susceptible animals, anthrax-infected animals, and anthrax-contaminated carcasses present in the soil. The presence of improperly disposed animal carcasses serves as a significant reservoir for potential infection. Ultimately, their model demonstrated that the Anthrax model's disease-free equilibrium is only locally stable when the fundamental reproduction number is smaller than one.

Additionally, by altering the animal contact rate, the Listeriosis-Anthrax coinfected population, the infected Anthrax population, and the infected Listeriosis population were all affected. This was done in order to recreate the Listeriosis-Anthrax coinfection model.

Anthrax is a serious and difficult infectious disease caused by the *Bacillus anthracis* bacteria, which releases spores that can live in the environment for years. The authors of [11] conducted research on pathogen identification and reservoirs to successfully control epidemics and sporadic disorders. One of the primary zoonotic food-borne infections, Listeria monocytogenes, is responsible for around 28% of all food-related deaths in the US each year and a large number of product recalls worldwide. Listeria monocytogenes has been demonstrated to exist on and within dairy farms. Models are often used in the study of the dynamics of infectious disease transmission. In recent years, there has been a notable increase in the use of mathematical models in the study of infectious diseases. Thus, a field known as mathematical epidemiology was born.

This study is based on the model developed by Kashyap et al. [4]. The original model was extended by incorporating additional compartments: A few days to many weeks may pass during the latent or incubation period of anthrax exposure. environment because anthrax spores are produced in soil by infected or terminal animals, and these spores break down in the environment and serve as a significant means of transmission. In the extended model, disease transmission occurs through two pathways: direct host-to-host transmission, represented by a standard incidence function, and indirect transmission through environmental spores, represented by a saturated environmental transmission function.

In order to analyze anthrax disease, the model put forth by [4]—which is frequently used for infectious diseases—has been modified to assume a path from susceptible to infected and recovered. However, this model ignores the anthrax's latent period, and the environment can play a significant role in anthrax transmission. A modified model was developed to overcome this limitation by incorporating exposed and environmental compartments. The effectiveness of several transmission methods (contact with contaminated animal products, spore inhalation, ingestion of contaminated food, contact with contaminated soil or animal products, etc.) can be examined with this improved model.

Recent extensions of compartmental epidemic models have incorporated additional transmission pathways and environmental factors to more realistically capture disease dynamics [12], motivating the inclusion of an environmental spore compartment in the present model. By incorporating these components, this study provides a better understanding of the dynamics of anthrax and transmission, guide the creation of evidence-based treatments, and assist in the creation of plans to encourage long-term recovery and lower the risk of anthrax in animals suffering from anthrax disease.

The paper is organized as follows: Section 2 contains my mathematical model analysis of anthrax disease. The model's equilibrium points and stability analysis are qualitatively analyzed in Section 3. In Section 4, my proposed model is subjected to numerical simulations. I provide a model simulation in order to enhance the systems and verify the analytical findings. Section 5 contains the study's conclusions.

2. Description and Formulation of the Model

In this section, this study formulates a deterministic mathematical model (Figure 1) of anthrax transmission dynamics, based on the following assumptions: susceptible animals to anthrax infection and designated by $S(t)$. Exposed animals which are infected but not yet infectious. Because anthrax have a latent period and denoted by $E(t)$. Infected animals are those showing anthrax symptoms and denoted by $I(t)$. Recovered animals are those recovered from anthrax infection and acquired temporal immunity and denoted by $R(t)$. Vaccinated represents

animals vaccinated against anthrax attack and denoted by $V(t)$. Environmental Spore concentration (non-host pathogen reservoir) and denoted by $B(t)$ with initial conditions $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, $V(0) \geq 0$, and $B(0) \geq 0$.

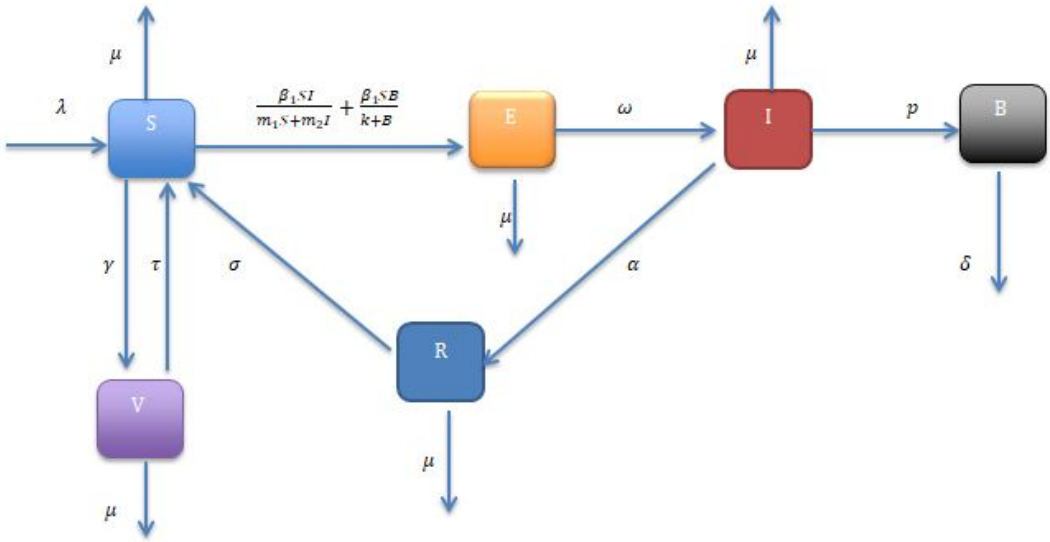


Figure 1. The flow chart of model (1).

$$\begin{aligned}
\frac{dS}{dt} &= \lambda - \frac{\beta_1 SI}{m_1 S + m_2 I} - \frac{\beta_2 SB}{k+B} - (\mu+\gamma)S + \sigma R + \tau V,\\
\frac{dE}{dt} &= \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k+B} - (\mu+\omega)E,\\
\frac{dI}{dt} &= \omega E - (\mu + p + \alpha)I,\\
\frac{dR}{dt} &= \alpha I - (\sigma+\mu)R,\\
\frac{dV}{dt} &= \gamma S - (\tau+\mu)V,\\
\frac{dB}{dt} &= pI - \delta B.
\end{aligned} \tag{1}$$

3. Analysis of the Model

3.1. Existence and Uniqueness of Solution

Theorem 1 (The existence and uniqueness of solution). *Let $\mathbb{D} = (S, E, I, R, V, B) \in \mathbb{R}^6_+$ denotes the region defined in ($|t - t_0| \leq k, ||y - y_0|| \leq n, y = (y_1, y_2, ..., y_6), y_0 = (y_{01}, y_{02}, ..., y_{0n})$) such that $||f(t, y_1) - f(t, y_2)|| \leq q||y_1 - y_2||$ and $1 \leq \epsilon \leq \mathbb{R}$ hold. Then the solution of the model system (1) is continuous and bounded by $\mathbb{D}$.*

Proof. Suppose $\mathbb{D}$ indicates the above region. It is sufficient to show $\frac{\partial f_i}{\partial y_j}$ $i, j = 1, 2, ..., 6$ are both continuous and bounded by $\mathbb{D}$. The region indicated contains

$$\mathbb{D} = (S, E, I, R, V, B).$$

Let

$$f_1 = \lambda - \frac{\beta_1 SI}{m_1 S + m_2 I} - \frac{\beta_2 SB}{k+B} - (\mu+\gamma)S + \sigma R + \tau V, \tag{2}$$

$$f_2 = \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k+B} - (\mu+\omega)E, \tag{3}$$

$$f_3 = \omega E - (\mu + p + \alpha)I, \tag{4}$$

$$f_4 = \alpha I - (\sigma+\mu)R, \tag{5}$$

$$f_5 = \gamma S - (\tau+\mu)V, \tag{6}$$

$$f_6 = pI - \delta B. \tag{7}$$

Equation system (1) be represented by f_1, f_2, f_3, f_4, f_5 and f_6 respectively then, based on ([13,14]), from the first three equations in the Equation system (1) I can get the following derivatives

$$\begin{array}{rcl} \left|\frac{\partial f_1}{\partial S}\right| & = & \left|-\frac{\beta_1 I(Sm_1+m_2I)+\beta_1 m_1 SI}{(Sm_1+m_2I)^2} - (\frac{\beta_2 B}{k+B} + \mu + \delta)\right| < \infty, \left|\frac{\partial f_1}{\partial E}\right| = |0| < \infty, \\ \left|\frac{\partial f_1}{\partial I}\right| & = & \left|-\frac{\beta_1 m_1 IS+\beta_1 m_2 SI^2-\beta_1 m_2 SI}{(Sm_1+m_2I)^2}\right| < \infty, \left|\frac{\partial f_1}{\partial R}\right| = |\delta| < \infty, \\ \left|\frac{\partial f_1}{\partial V}\right| & = & |\tau| < \infty, \left|\frac{\partial f_1}{\partial B}\right| = |-\frac{\beta_2 kS}{(k+B)^2}| < \infty \end{array}$$

Similarly, the same method is applied to f_2, f_3, f_4, f_5 and f_6. The above partial derivatives are exist, continuous and bounded. Consequently, the system of Equation (1) is differentiable for each of the variables and each of its partial derivatives is finite and continuous. Therefor, the solution of system Equation (1) exists and is unique to the region $\mathbb{D}$. □

3.2. The Solution's Boundedness

Theorem 2 (**Boundedness for host population**). *All the solutions $S(t)$, $E(t)$, $I(t)$, $R(t)$, $V(t)$ of the model (1) are bounded.*

Proof. The objective is to demonstrate that the total host population size of the entire system, $N_h(t)$, is bounded in order to demonstrate that the population size of each compartment is bounded. The population as a whole is determined by:

$$\begin{array}{rcl} N_h(t) & = & S(t) + E(t) + I(t) + R(t) + V(t) \\ \frac{dN_h(t)}{dt} & = & \lambda - \mu N_(t) - pI - \alpha I \\ \frac{dN_h(t)}{dt} & \leq & \lambda - \mu N_h(t) \end{array} \quad (8)$$

Rearranging (8) produces

$$\frac{dN_h(t)}{dt} + \mu N(t) \leq \lambda \quad (9)$$

$e^{\mu t}$ is obtained by finding the integrating factor for the linear first-order differential Equation (9). Upon multiplying and integrating both sides of Equation (9) by $e^{\mu t}$, I get

$$N_h(t) \leq \frac{\lambda}{\mu} + (N_h(0) - \frac{\lambda}{\mu})e^{-\mu t} \quad (10)$$

As $t \to \infty$, then $N_h(t) \leq max\{N_h(0), \frac{\lambda}{\mu}\} = M_h$.
Therefore, all host compartments are bounded: $S(t), E(t), I(t), R(t), V(t) \leq M_h$. □

Theorem 3 (**Boundedness for the Environmental Reservoir $B(t)$**). *The model (1) of the solution $B(t)$ is bounded.*

Proof. After establishing the strict upper bound $I(t) \leq M_h$, the standalone differential equation for the pathogen reservoir can be considered.

$$\frac{B(t)}{dt} = pI(t) - \delta B(t)$$

Since $I(t) \leq M_h$ for $t \geq 0$

$$\begin{array}{rcl} \frac{B(t)}{dt} & \leq & pM_h(t) - \delta B(t) \\ \frac{B(t)}{dt} + \delta B(t) & \leq & pM_h(t) \end{array}$$

The integrating factor is $e^{\delta t}$.

$$B(t) \leq \frac{p}{\delta} M_h + (B(0) - \frac{p}{\delta} M_h)e^{-\delta t} \quad (11)$$

As $t \to \infty$, the exponential tern vanishes then $\lim_{t\to\infty} SupB(t) \frac{p}{\delta} M_h$. □

Finlay, from Theorems 2 and 3 for all times t, the feasible solution set of the model system (1) stays in the region $\Omega = \{(S, E, I, R, V, B) \in \mathbb{R}^6_+; 0 \leq S+E+I+V+R \leq max\{N_h(0), \frac{\lambda}{\mu}\}, 0 \leq B \leq \frac{p}{\delta}(max\{N_h(0), \frac{\lambda}{\mu}\})$.

Therefore, the region is positively invariant for all solutions with condition to $\mathbb{R}^6_+$.

3.3. The Solution's Positivity

Theorem 4 (Positivity). *The solution $S(t)$, $E(t)$, $I(t)$, $R(t)$, $V(t)$, $B(t)$ of the model system (1) are non-negative $\forall t \geq 0$ if the initial conditions of the system are $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, $V(0) \geq 0$, $B(0) \geq 0$.*

Proof. Following the methodology described in [14–16], the non-negativity of the solutions to model (1) is demonstrated. Thus, based on the first equation's model system (1), I have

$$\frac{dS}{dt} = \lambda - \frac{\beta_1 SI}{m_1 S + m_2 I} - \frac{\beta_2 SB}{k+B} + (\mu+\gamma)S + \sigma R + \tau V \tag{12}$$

where

$$\Lambda_1 = \frac{\beta_1 I}{m_1 S + m_2 I} + \frac{\beta_2 B}{k+B}.$$

Re-arranging (12) provides

$$\frac{dS}{dt} + (\mu + \Lambda_1 + \gamma)S \geq \lambda. \tag{13}$$

For the state variable S, Equation (13) is a separable first-order differential equation. Determining the integrating factor results in $e^{\int(\mu+\Lambda_1+\gamma)dt}$, I obtain

$$S(t) \geq e^{-(\mu+\Lambda_1+\gamma)t}(\lambda \int e^{(\mu+\Lambda_1+\gamma)t}dt). \tag{14}$$

Following integration and simplification (14), I obtain

$$S(t) \geq e^{-(\mu+\Lambda_1+\gamma)t}K_1 + \frac{\lambda}{\mu+\Lambda_1+\gamma}. \tag{15}$$

where K_1 is constant.

The following value of K_1 is obtained by computing (15) at $t = 0$

$$K_1 \geq S(0) - \frac{\lambda}{\mu+\Lambda_1+\gamma} \tag{16}$$

Replacing (16) to (15) I acquire

$$S(t) \geq S(0)e^{-(\mu+\Lambda_1+\gamma)t} + \frac{\lambda}{\mu+\Lambda_1+\gamma}(1 - e^{-(\mu+\Lambda_1+\gamma)t}). \tag{17}$$

Since $S(0) > 0$, $e^{-(\mu+\Lambda_1+\gamma)t} > 0$ and $\frac{\lambda}{\mu+\Lambda_1+\gamma} > 0\ \forall t \geq 0$. Therefore,

$$S(t) \geq S(0)e^{-(\mu+\Lambda_1+\gamma)t} + \frac{\lambda}{\mu+\Lambda_1+\gamma}(1 - e^{-(\mu+\lambda_1+\gamma)t}) > 0.$$

Using the same method for $E(t), I(t), R(t), V(t)$, and $B(t)$, I get

$$\begin{aligned}
E(t) &= E(0)e^{-(\omega+\mu)t}\frac{\Lambda_1}{\mu+\omega}(1 - e^{-(\mu+\omega)t}) \geq 0.\\
I(t) &= I(0)e^{-(\alpha+p+\mu)t} + \frac{\omega}{\alpha+p++\mu}E(1 - e^{-(\alpha+p++\mu)t}) \geq 0.\\
R(t) &= R(0)e^{-(\sigma+\mu)t} + \frac{\alpha}{\sigma+\mu}I(1 - e^{-(\sigma+\mu)t}) \geq 0.\\
V(t) &= V(0)e^{-(\tau+\mu)t} + \frac{\gamma}{\tau+\mu}S(1 - e^{-(\tau+\mu)t}) \geq 0.\\
B(t) &= B(0)e^{-\delta t} + \frac{p}{\delta}I(1 - e^{-\delta t}) \geq 0.
\end{aligned}$$

As a result, for all $t \geq 0$, the solutions $S(t), E(t), I(t), R(t), V(t)$, and $B(t)$ in the system (1) stay non-negative. □

3.4. Anthrax Free Equilibrium (AFE) Point

Assuming the absence of anthrax, the initial conditions are taken as $E^0 = 0$, $I^0 = 0$, $I^0 = 0$, $R^0 = 0$, and $B^0 = 0$. Accordingly, the model's anthrax free equilibrium point (1) is provided by

$$E_0 = (S^0, E^0, I^0, R^0, V^0, B^0) = (\frac{\lambda}{\gamma+\mu}, 0, 0, 0, \frac{\gamma\lambda}{(\gamma+\mu)(\tau+\mu)}, 0).$$

3.5. The Basic Reproduction Number

The basic reproduction number (R_0) is the average number of secondary infections that arise from the introduction of a single infection into a susceptible population. Following the next-generation matrix approach presented in [17], the basic reproduction number R_0 is calculated. The elements of the next generation matrix, F and V, represent the emergence of new infections and the movement of infected individuals from one compartment to another, respectively.

Anthrax disease compartments are now as follows, based on ([14,17,18]) from the model system (1):

$$\begin{aligned} E' &= \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k+B} - (\mu+\omega)E \\ I' &= \omega E - (\mu + p + \alpha)I \\ B' &= pI - \delta B \end{aligned}$$

$$f_i = \begin{bmatrix} \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k+B} \\ 0 \\ 0 \end{bmatrix}; v_i = \begin{bmatrix} (\mu+\omega)E \\ -\omega E + (\mu + p + \alpha)I \\ -pI + \delta B \end{bmatrix}$$

At E_0, the Jacobian matrix of f_i and v_i is expressed as

$$F = \begin{bmatrix} 0 & \frac{\beta_1}{m_1} & \frac{\beta_1 S^0}{k} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}; V = \begin{bmatrix} a_1 & 0 & 0 \\ -\omega & a_2 & 0 \\ 0 & -p & \delta \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{a_1} & 0 & 0 \\ \frac{\omega}{a_1 a_2} & \frac{1}{a_2} & -\frac{1}{a_2} \\ \frac{\omega p}{a_1 a_2 \delta} & \frac{p}{a_2 \delta} & \frac{1}{\delta} \end{bmatrix}$$

where

$$a_1 = \omega + \mu, a_2 = p + \alpha + \mu$$

Thus, the spectrum radius of FV^{-1}, which is determined as follows, is the basic reproduction number

$$R_0 = \frac{\omega}{(\mu + p + \alpha)(\mu + \omega)}(\frac{\beta_1}{m_1} + \frac{\beta_2 p\lambda}{k\delta(\mu+\gamma)}) \tag{18}$$

3.6. Local Stability of AFE

Theorem 5. *If $R_0 < 1$, the system (1)'s anthrax free equilibrium point E_0 is locally asymptotically stable.*

Proof. At the anthrax free equilibrium point E_0, the Jacobian matrix of the system (1) is provided by

$$J(E_0) = \begin{bmatrix} -a_3 & 0 & \frac{-\beta_1}{m_1} & \sigma & \tau & -\frac{\beta_2\lambda}{ka_3} \\ 0 & -a_1 & \frac{\beta_1}{m_1} & 0 & 0 & \frac{\beta_2\lambda}{ka_3} \\ 0 & \omega & -a_2 & 0 & 0 & 0 \\ 0 & 0 & \alpha & -a_4 & 0 & 0 \\ \gamma & 0 & 0 & 0 & -a_5 & 0 \\ 0 & 0 & p & 0 & 0 & -\delta \end{bmatrix}$$

Thus, the characteristics equation of $J_6(E_0)$ is given by

$|J_6(E_0) - \lambda I_6| = 0$, where I_6 is a 6 by 6 identity square matrix.

$$(k+\lambda)(c_5\lambda^5 + c_4\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0) = 0$$

Clearly, the first eigenvalue is $\lambda_1 = -k$ and the remaining eigenvalues $\lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ can be derived from the reduced equation that follows:

$$c_5\lambda^5 + c_4\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0$$

where

$$\begin{aligned}
c_0 &= \frac{\beta_1}{m_1}\tau\delta\gamma + 2a_3a_1a_2a_5\delta + \tau\frac{\lambda\beta_2}{a_3k}\omega p\gamma - a_3\frac{\beta_1}{m_1}a_5\delta - a_3\frac{\lambda\beta_2}{a_3k}\omega a_5 p - 2\tau a_1a_2\delta\gamma \\
c_1 &= 2a_1a_2a_5\delta + a_3a_1a_5\delta + a_3a_2a_5\delta + a_3a_1a_2a_5 + \frac{\beta_1}{m_1}\tau\omega\gamma + 2a_3a_1a_2\delta \\
&\quad - \tau a_1a_2\gamma - a_3\frac{\beta_1}{m_1}\delta - \frac{\beta_1}{m_1}a_5\delta - a_3\frac{\beta_1}{m_1}\omega a_5 - a_3\frac{\lambda\beta_2}{a_3k}\omega p - \frac{\lambda\beta_2}{a_3k}\omega p a_5 - \tau a_1\delta\gamma - \tau a_2\delta\gamma \\
c_2 &= a_1a_2a_5 + a_3a_1a_2 + a_3a_1a_5 + a_3a_2a_5 + a_3a_1\delta + a_3a_2\delta + a_3a_5q + 2a_1a_2\delta + a_1a_5\delta + a_2a_5\delta - \frac{\beta_1}{m_1}\delta \\
&\quad - a_3\frac{\beta_1}{m_1}\omega - \frac{\lambda\beta_2}{ka_3}\omega p - \tau a_1\alpha - \tau - \frac{\beta_1}{m_1}\omega a_5a_2\alpha - \tau\delta\gamma \\
c_3 &= a_1\delta + a_3a5 + a_1a_2 + a_3\delta + a_1a_5 + a_2a_5 + a_2\delta + a_5\delta + a_3a_1 + a_3a_2 - \frac{\beta_1}{m_1}\omega - \tau\gamma \\
c_4 &= a_3 + a_1 + a_2 + a_5 \\
c_5 &= 1 \\
a_3 &= \mu + \gamma \\
a_4 &= \delta + \mu \\
a_5 &= \tau + \mu
\end{aligned}$$

If $c_4 > 0, c_3 > 0, c_2 > 0, c_1 > 0, c_0 > 0$, $c_4c_3c_2 > c_2^2 + c_4^2c_1$ and $(c_4c_1 - c_0)(c_4c_3c_2 - c_2^2 - c_4^2c_1) > c_0(c_4c_3 - c_2)^2 + c_4c_0^2$, then all of the characteristic equation's eigenvalues have a negative real part, according to the Routh-Hurwitz criteria. Since all of the parameters are positive and their total is positive, I can see that c_4 is positive. However, to state that c_3, c_2,c_1 and c_0 are positive $a_1\delta + a_3a5 + a_1a_2 + a_3\delta + a_1a_5 + a_2a_5 + a_2\delta + a_5\delta + a_3a_1 + a_3a_2 - (\frac{\beta_1}{m_1}\omega\tau\gamma)$, $a_1a_2a_5 + a_3a_1a_2 + a_3a_1a_5 + a_3a_2a_5 + a_3a_1\delta + a_3a_2\delta + a_3a_5q + 2a_1a_2\delta + a_1a_5\delta + a_2a_5\delta - (\frac{\beta_1}{m_1}\delta + a_3\frac{\beta_1}{m_1}\omega + \frac{\lambda\beta_2}{ka_3}\omega p + \tau a_1\alpha + \tau\frac{\beta_1}{m_1}\omega + a_5a_2\alpha + \tau\delta\gamma)$,$2a_1a_2a_5\delta + a_3a_1a_5\delta + a_3a_2a_5\delta + a_3a_1a_2a_5 + \frac{\beta_1}{m_1}\tau\omega\gamma + 2a_3a_1a_2\delta - (\tau a_1a_2\gamma + a_3\frac{\beta_1}{m_1}\delta + \frac{\beta_1}{m_1}a_5\delta + a_3\frac{\beta_1}{m_1}\omega a_5 + a_3\frac{\lambda\beta_2}{a_3k}\omega p + \frac{\lambda\beta_2}{a_3k}\omega p a_5 + \tau a_1\delta\gamma + \tau a_2\delta\gamma)$ and $\frac{\beta_1}{m_1}\tau\delta\gamma + 2a_3a_1a_2a_5\delta + \tau\frac{\lambda\beta_2}{a_3k}\omega p\gamma - (a_3\frac{\beta_1}{m_1}a_5\delta + a_3\frac{\lambda\beta_2}{a_3k}\omega a_5 p + 2\tau a_1a_2\delta\gamma)$ must be positive, respectively. Hence, $c_3 > 0$ if it fulfills that $a_1\delta + a_3a5 + a_1a_2 + a_3\delta + a_1a_5 + a_2a_5 + a_2\delta + a_5\delta + a_3a_1 + a_3a_2 > \frac{\beta_1}{m_1}\omega\tau\gamma$, $c_2 > 0$ if it fulfills that $a_1a_2a_5 + a_3a_1a_2 + a_3a_1a_5 + a_3a_2a_5 + a_3a_1\delta + a_3a_2\delta + a_3a_5q + 2a_1a_2\delta + a_1a_5\delta + a_2a_5\delta > (\frac{\beta_1}{m_1}\delta + a_3\frac{\beta_1}{m_1}\omega + \frac{\lambda\beta_2}{ka_3}\omega p + \tau a_1\alpha + \tau\frac{\beta_1}{m_1}\omega + a_5a_2\alpha + \tau\delta\gamma$,$c_1 > 0$ if it fulfills that $2a_1a_2a_5\delta + a_3a_1a_5\delta + a_3a_2a_5\delta + a_3a_1a_2a_5 + \frac{\beta_1}{m_1}\tau\omega\gamma + 2a_3a_1a_2\delta > \tau a_1a_2\gamma + a_3\frac{\beta_1}{m_1}\delta + \frac{\beta_1}{m_1}a_5\delta + a_3\frac{\beta_1}{m_1}\omega a_5 + a_3\frac{\lambda\beta_2}{a_3k}\omega p + \frac{\lambda\beta_2}{a_3k}\omega p a_5 + \tau a_1\delta\gamma + \tau a_2\delta\gamma$ and $c_0 > 0$ if it fulfills that $\frac{\beta_1}{m_1}\tau\delta\gamma + 2a_3a_1a_2a_5\delta + \tau\frac{\lambda\beta_2}{a_3k}\omega p\gamma > a_3\frac{\beta_1}{m_1}a_5\delta + a_3\frac{\lambda\beta_2}{a_3k}\omega a_5 p + 2\tau a_1a_2\delta\gamma$. Additionally, $c_4c_3c_2 > c_2^2 - c_4^2c_1$ and $(c_4c_1 - c_0)(c_4c_3c_2 - c_2^2 - c_4^2c_1)$. From the Routh-Hurwitz criterion, it follows that $c_4c_3c_2 > c_2^2 - c_4^2c_1$ if $c_1 = 2a_1a_2a_5\delta + a_3a_1a_5\delta + a_3a_2a_5\delta + a_3a_1a_2a_5 + \frac{\beta_1}{m_1}\tau\omega\gamma + 2a_3a_1a_2\delta - \tau a_1a_2\gamma - a_3\frac{\beta_1}{m_1}\delta - \frac{\beta_1}{m_1}a_5\delta - a_3\frac{\beta_1}{m_1}\omega a_5 - a_3\frac{\lambda\beta_2}{a_3k}\omega p - \frac{\lambda\beta_2}{a_3k}\omega p a_5 - \tau a_1\delta\gamma - \tau a_2\delta\gamma > 0$ and $(c_4c_1 - c_0)(c_4c_3c_2 - c_2^2 - c_4^2c_1) > c_0(c_4c_3 - c_2)^2 + c_4c_0^2$ while $c_4c_1 > c_0$, $c_4c_3c_2 > c_2^2 + c_4^2c_1$ for $R_0 < 1$.

Therefore, if $R_0 < 1$, then all of the eigenvalues of the characteristic equation are obviously real and negative. If $R_0 < 1$, I can deduce that the system's anthrax equilibrium point (1) is locally asymptotically stable. □

3.7. Global Stability of AFE

Theorem 6. *If $S^0 \geq S$, the anthrax free equilibrium E_0 of the system (1) is globally asymptotically stable for $R_0 < 1$.*

Proof. Let's change the system

$$\begin{aligned}
\tfrac{dZ_1}{dt} &= F(Z_1, Z_2) \\
\tfrac{dZ_2}{dt} &= G(Z_1, Z_2), \quad G(Z_1, 0) = 0
\end{aligned}$$

where $Z_1 = (S, R, V) \in \mathbb{R}^3_+$ is the group of animals that do not have anthrax and $Z_2 = (E, I, B) \in \mathbb{R}^3_+$ denotes the category of illness that exists. $U_0 = (Z_1^*, 0)$ represents the AFE point of the model, where $Z_1^* = (\frac{\Lambda}{\mu}, 0, \frac{\gamma\lambda}{(\gamma+\mu)(\tau+\mu)})$. In the Theorem 5, I proved that E_0 is locally asymptotically stable. I demonstrate global stability using the Castillo-Chavez theorem, using the methods of Iyasu and Endiriyas [14]. The system (1) provides me with

$$\frac{dZ_1}{dt} = F(Z_1, Z_2) = \begin{bmatrix} \lambda - \frac{\beta_1 SI}{m_1 S + m_2 I} - \frac{\beta_2 SB}{k+B} - (\mu+\gamma)S + \delta R + \tau V \\ \alpha I - (\sigma+\mu)R \\ \gamma S - (\tau+\mu)V \end{bmatrix}$$

$$\frac{dZ_1}{dt} = G(Z_1, Z_2) = \begin{bmatrix} \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k+B} - (mu+\omega)E \\ \omega E - (\mu + p + \alpha)I \\ pI - \delta B \end{bmatrix}$$

I To show that Z_1^* is globally asymptotically stable for the system $\frac{dZ_1}{dt} = F(Z_1, 0)$, I enter $E = 0, I = 0$, and $B = 0$ to produce the simplified system.

$$\frac{dZ_1}{dt} = F(Z_1, 0) = \begin{bmatrix} \lambda - (\mu+\gamma)S + \delta R + \tau V \\ -(\sigma+\mu)R \\ \gamma S - (\tau+\mu)V \end{bmatrix}$$

I get new system

$$\frac{dS(t)}{dt} = \lambda - (\mu+\gamma)S + \delta R + \tau V \tag{19a}$$

$$\frac{dR(t)}{dt} = -(\sigma+\mu)R \tag{19b}$$

$$\frac{dV(t)}{dt} = \gamma S - (\tau+\mu)V \tag{19c}$$

The system (19) represents a non-homogeneous linear system of ordinary differential equations. Equations (19a)–(19c) are solved to provide the following solutions.

$$S(t) \geq S(0)e^{-(\mu+\Lambda_1+\gamma)t} + \frac{\lambda}{\mu+\Lambda_1+\gamma}(1 - e^{-(\mu+\lambda_1+\gamma)t})$$

$$R(t) = R(0)e^{-(\sigma+\mu)t} + \frac{\alpha}{\sigma+\mu}I(t)(1 - e^{-(\sigma+\mu)t})$$

$$V(t) = V(0)e^{-(\tau+\mu)t} + \frac{\gamma}{\tau+\mu}S(t)(1 - e^{-(\tau+\mu)t})$$

Taking as $t \to \infty$, I obtain

$$\lim_{t\to\infty}(S(t), R(t), V(t)) = (\frac{\lambda}{\gamma+\mu}, 0, \frac{\gamma\lambda}{(\gamma+\mu)(\tau+\mu)}) = Z_1^*.$$

Therefore, Z_1^* is globally asymptotically stable to the system $\frac{dZ_1}{dt} = F(Z_1, 0)$.

II I will demonstrate that $G(Z_1, Z_2) = MZ_2 - \hat{G}(Z_1, Z_2)$ and $\hat{G}(Z_1, Z_2) \geq 0$ for every $(Z_1, Z_2) \in \Omega$, where $M = \frac{\partial G(Z_1^*, 0)}{\partial Z_2}$ is a Metzler matrix with non-negative off-diagonal elements and Ω is the region jwhere the model makes biological sense.

Consider the matrix

$$M = \frac{\partial G(Z_1^*, 0)}{\partial Z_2}$$

$$M = \begin{bmatrix} -(\omega+\mu) & \frac{\beta_1}{m_1^2} & \frac{\beta_2 S(0)}{k} \\ \omega & -(\alpha+p+\mu) & 0 \\ 0 & p & -\delta \end{bmatrix}$$

Hence, M is a Metzer matrix (off diagonal elements are non-negative). Here,

$$\hat{G}(Z_1, Z_2) = MZ_2 - G(Z_1, Z_2)$$

After performing some simplifications, I obtain

$$\hat{G}(Z_1, Z_2) = \begin{bmatrix} \frac{\beta_1 I S^0}{m_1 S + m_2 I} + \frac{\beta_2 B S^0}{k+B} - \frac{\beta_1 I S}{m_1 S + m_2 I} - \frac{\beta_2 B S}{k+B} \\ 0 \\ 0 \end{bmatrix}$$

This implies that

$$\hat{G}(Z_1, Z_2) = (S^0 - S) \begin{bmatrix} \frac{\beta_1 I}{m_1 S + m_2 I} + \frac{\beta_2 B}{k+B} \\ 0 \\ 0 \end{bmatrix}$$

Since $S \leq S^0$, I have $(S^0 - S)(\frac{\beta_1 I}{m_1 S + m_2 I} + \frac{\beta_2 B}{k+B}) \geq 0$; the preceding matrix is non-negative. Therefore, it can be written as

$$\hat{G}(Z_1, Z_2) = (S^0 - S) \begin{bmatrix} \frac{\beta_1 I}{m_1 S + m_2 I} + \frac{\beta_2 B}{k+B} \\ 0 \\ 0 \\ 0 \end{bmatrix} \geq 0$$

Therefore, the anthrax free equilibrium point E^0 is globally asymptotically stable for $R_0 < 1$ by the Castillo-Chavez theorem. □

3.8. Endemic Equilibrium (EE) Point

There is an equilibrium point known as the endemic equilibrium point, which appears by the existence of anthrax disease in the population denoted by $E_1 = (S^*, E^*, I^*, R^*, V^*, B*)$. It can be derived by setting each system Equation (1) to zero, according to the method of Iyasu and Endiriyas [14]:

$$\lambda - \frac{\beta_1 S I}{m_1 S + m_2 I} - \frac{\beta_2 S B}{k+B} - (\mu + \gamma) S + \sigma R + \tau V = 0 \tag{20a}$$

$$\frac{\beta_1 S I}{m_1 S + m_2 I} + \frac{\beta_2 S B}{k+B} - (\mu + \omega) E = 0 \tag{20b}$$

$$\omega E - (\mu + p + \alpha) I = 0 \tag{20c}$$

$$\alpha I - (\sigma + \mu) R = 0 \tag{20d}$$

$$\gamma S - (\tau + \mu) V = 0 \tag{20e}$$

$$pI - \delta B = 0 \tag{20f}$$

From Equation (20f), I have

$$B^* = \frac{pI^*}{\delta} \tag{21}$$

From Equation (20e), I have

$$V^* = \frac{\gamma S^*}{\tau + \mu} \tag{22}$$

From Equation (20d), I have

$$R^* = \frac{\alpha S^*}{\delta + \mu} \tag{23}$$

From Equation (20c), I have

$$E^* = \frac{(\alpha + p + \mu) I^*}{\omega} \tag{24}$$

By inserting Equations (21) and (24) to the Equation (20b), I obtain

$$S^* = \frac{-(\beta_1 I^* + m_2 D I * - C m_1) \pm \sqrt{(\beta_1 I^* + m_2 D I^* - C m_1)^2 + 4 D m_1 m_2 C I^*}}{2 D m_1} \tag{25}$$

where

$$C = \frac{(\mu + \omega)(\mu + \alpha + p)}{\omega} I^*$$

$$D = \frac{\beta_2 p I^*}{k\delta + p I^*}$$

$S^* > 0$, I take biologically admissible positive root such that $S^*(I^*) > 0$.

By inserting Equations (21)–(23) to the Equation (20a) and doing some simplification, I get

$$-\frac{\beta_1 S^* I^*}{m_1 S^* + m_2 I^*} - \frac{\beta_2 S^* B^*}{k + B^*} + \frac{\delta \alpha I^*}{\delta + \mu} - (S^*((\mu + \gamma) - \frac{\tau\gamma}{\tau + \mu}) - \lambda) = 0 \tag{26}$$

So that $I^* > 0$ is any positive solution (for biological meaning) that makes $S^*(I^*) > 0$, gives an endemic equilibrium. Therefore, the endemic equilibrium point is given by

$$E_1 = (S^*, E^*, I^*, R^*, V^*, B^*)$$

where

$$\begin{aligned} E^* &= \frac{(\alpha + p + \mu)I^*}{\omega} \\ R^* &= \frac{\alpha S^*}{\delta + \mu} \\ V^* &= \frac{\gamma S^*}{\tau + \mu} \\ B^* &= \frac{pI^*}{\delta} \end{aligned}$$

This indicates that if $R_0 > 1$, the endemic equilibrium is present. Additionally, the force of infection can be expressed as

$$\Lambda^* = \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{k + B}. \tag{27}$$

3.9. Local Stability of EE

Theorem 7. *If $R_0 > 1$, the endemic equilibrium point of model (1) is locally asymptotically stable; if $R_0 < 1$, it is unstable.*

Proof. In order to investigate local stability at the dynamical system's endemic equilibrium point (E_1), following the methodology presented in [14, 19], the Jacobian matrix of system (1) is evaluated at the endemic equilibrium point E_1. The result is provided by

$$J(E_1) = \begin{bmatrix} -q - a_3 & 0 & -r & \sigma & \tau & -t \\ q & -a_1 & r & 0 & 0 & t \\ 0 & \omega & -a_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_4 & 0 & 0 \\ \gamma & 0 & 0 & 0 & -a_5 & 0 \\ 0 & 0 & p & 0 & 0 & -\delta \end{bmatrix}$$

where

$$\begin{aligned} q &= \frac{\beta_1^*(m_1 S^* + m_2 I^*) - \beta_1 m_1 S^* I^*}{(m_1 S^* + m_2 I^*)^2} + \frac{\beta_2 B^*}{K + B^*} \\ r &= \frac{\beta_1^*(m_1 S^* + m_2 I^*) - \beta_1 m_1 S^* I^*}{(m_1 S^* + m_2 I^*)^2} \\ t &= \frac{\beta_2 B^*(k + B^*) - \beta_2 S^* B^*}{(k + B^*)^2} \end{aligned}$$

From $|J(E_1) - \lambda I_6| = 0$, where I_6 is a 6 by 6 identity matrix, I deduce the characteristics equation of $J(E_1)$.

$$p(\lambda) = 0$$
$$\lambda^6 + c_1\lambda^5 + c_2\lambda^4 + m_3\lambda^3 + c_4\lambda^2 + c_5\lambda + c_6 = 0$$

where

$$
\begin{aligned}
c_1 &= a_3 + a_1 + a_2 + a_4 + \delta + a_+ q \\
c_2 &= a_3a_1 + a_3a_2 + a_1a_2 + a_3 + a_1\delta + a_3a_5 + a_2\delta + a_1a_5 + a_2a_5 + \delta a_5 \\
&\quad + a_1q - \gamma\tau - \omega r + a_2q + \delta q + a_5q + a_4(a_3 + a_1 + a_1 + \delta + a_5 + q) \\
c_3 &= a_4(a_3a_1 + a_3a_2 + \omega + a_3\delta + a_1\delta + a_3a_5 + a_2\delta + a_1a_5 + a_2a_5 + \delta a_5 + a_1q - \gamma m - \omega r + a_2q + \delta q \\
&\quad + a_5q) + a_3a_1a_2 + a_3a_1\delta + a_5a_2\delta + a_3a_1a_5 + a_1a_2\delta + a_3a_2a_5 + a_1a_2a_5 + a_1\delta a_5 - a_5\omega r - a_1\gamma\tau \\
&\quad + a_2\delta a_5 + a_1a_2q - a_2\gamma\tau + a_1\delta q - \gamma\delta\tau - \omega\delta r + a_2\delta q + a_1a_5q - \omega a_5r + a_2a_5q + \delta a_5q - \omega pt \\
c_4 &= a_4(a_3a_1a_2 + a_3a_1\delta a_3a_2\delta + a_3a_1a_5 + a_1a_2\delta + a_3a_2a_5 + a_1a_2a_5 + a_3\delta a_5a_1\delta a_5 - a_3\omega r - a_1\gamma\tau \\
&\quad + a_2\delta a_5 + a_1a_2q - a_2\gamma\tau + a_1\delta q - \gamma\delta\tau - \omega\delta r + a_2\delta q + a_1a_5q - \omega a_5r + a_2a_5q + \delta a_5q - \omega pt) \\
&\quad + a_1a_2\delta q - a_2\gamma\delta\tau - a_2\gamma\delta\tau - a_3\omega a_5r + a_1a_2a_5q + a_1\delta a_5q - a_3\omega pt - \omega\delta a_5r + a_2\delta a_5q + \omega\gamma\tau r - \omega a_5pt \\
&\quad + a_3a_1a_2\delta + a_3a_1a_2a_5 + a_3a_1\delta a_5 + a_3a_2\delta a_5 + a_1a_2\delta a_5 - a_1a_2\gamma\tau - a_3\omega\delta r - a_1\gamma\delta\tau \\
c_5 &= a_4(a_1a_2\delta q - \omega\gamma\delta\tau - a_3\omega a_5r + a_1a_2a_5q + a_1\delta a_5q - a_3\omega pt - \omega\delta a_5r + a_2\delta a_5q + \omega\gamma\tau r - \omega a_5pt \\
&\quad + a_3a_1\omega\delta + a_3a_1a_2a_5 + a_3a_1\delta a_5 + a_1a_2\delta a_5 - a_1a_2\gamma\tau - a_3\omega\delta r - a_1\gamma\delta\tau) + a_3a_1a2\delta a_5 - \\
&\quad a_1a_2\gamma\delta\tau - a_3\omega\delta a_5r + a_1a_2\delta a_5q + \omega\gamma\delta\tau r - a_3\omega a_5pt + \omega\gamma\tau pt \\
c_6 &= a_4(a_3a_1a_2\delta a_5 - a_1a_2\gamma\delta\tau - a_3\omega\delta a_5r + a_1a_2\delta a_5q + \delta\gamma\delta\tau r - a_3\omega a_5pt + \omega\gamma\tau pt)
\end{aligned}
$$

The characteristics polynomial equation is as follows:

$$
\begin{aligned}
p(\lambda) &= 0 \\
\lambda^6 + c_1\lambda^5 + c_2\lambda^4 + m_3\lambda^3 + c_4\lambda^2 + c_5\lambda + c_6 &= 0
\end{aligned}
$$

the following is the alternate Routh Hurwitz array:

$$
\begin{array}{c|cccc}
\lambda^6 & 1 & c_2 & c_4 & c_6 \\
\lambda^5 & c_1 & c_3 & c_5 & 0 \\
\lambda^4 & b_1 & b_2 & b_3 & 0 \\
\lambda^3 & m_1 & m_2 & 0 & \\
\lambda^2 & d_1 & d_2 & & \\
\lambda^1 & e_1 & 0 & & \\
\lambda^0 & c_0 & & &
\end{array}
$$

where

$$
\begin{aligned}
b_1 &= \frac{c_1c_2 - c_3 \times 1}{c_1} \qquad b_2 = \frac{c_1c_4 - c_5 \times 1}{c_1} \\
b_3 &= \frac{c_1c_6 - 0 \times 1}{c_1} \qquad m_1 = \frac{b_1c_3 - c_1b_2}{b_1} \\
m_2 &= \frac{b_1c_5 - b_3c_1}{b_1} \qquad d_1 = \frac{m_1b_2 - b_1m_2}{m_1} \\
d_2 &= \frac{m_1b_3 - 0 \times b_2}{m_1} \qquad e_1 = \frac{d_2m_2 - d_2m_1}{d_1}
\end{aligned}
$$

Next, I have two requirements that show E_1 local stability using the Routh-Hurwitz criteria:

(i) When all the elements in the first column of the Routh-Hurwitz array have the same algebraic sign, I am sure that the population has anthrax and that the endemic equilibrium point is stable.

(ii) If each element in the first column has a separate algebraic sign, the endemic equilibrium point of the Routh-Hurwitz array is unstable, indicating the lack of anthrax in the population. □

3.10. Global Stability of EE

Theorem 8. The system's endemic equilibrium point is globally asymptotically stable on Ω if $R_0 > 1$.

Proof. To prove that the endemic equilibrium point E_1, is globally asymptotically stable, the following Lyapunov function is considered.

$$
\begin{aligned}
&L(S^*, E^*, I^*, R^*, V^*, B^*) \\
&= S - S^* + S^* \times ln(\frac{S^*}{S}) \\
&+ E - E^* + E^* \times ln(\frac{E^*}{E}) \\
&+ I - I^* + I^* \times ln(\frac{I^*}{I}) \\
&+ R - R^* + R^* \times ln(\frac{R^*}{R}) \\
&+ V - V^* + V^* \times ln(\frac{V^*}{V}) \\
&+ B - B^* + B^* \times ln(\frac{B^*}{B})
\end{aligned} \tag{28}
$$

Differentiating (28) I obtain:

$$
\begin{aligned}
\frac{dL}{dt} &= \frac{S - S^*}{S}\frac{dS}{dt} + \frac{E - E^*}{E}\frac{dE}{dt} + \frac{I - I^*}{I}\frac{dI}{dt} \\
&+ \frac{R - R^*}{R}\frac{dR}{dt} + \frac{V - V^*}{V}\frac{dV}{dt} + \frac{B - B^*}{B}\frac{dB}{dt}
\end{aligned} \tag{29}
$$

By substituting (1) and simplifying (29) I get

$$
\frac{dL}{dt} = P - M \tag{30}
$$

where P and M are the positive and negative terms of (30, respectively). That is

$$
\begin{aligned}
P &= \lambda + (S^* + S)(\frac{\beta_1 I}{m_1 S + m_2 I}) + \gamma S + S^*(\mu + \gamma) + (\delta + \alpha)R + \tau V + (tau + \mu)V^* \\
&+ (\mu + \omega)E^* + \omega E + (\mu + p + \alpha)I^* + \alpha I + (\delta\mu)R^*
\end{aligned}
$$

$$
\begin{aligned}
M &= \frac{\beta_1 SI}{m_1 S + m_2 I} + \frac{\beta_2 SB}{K + B} + (\mu + \gamma)S + (\lambda + \delta R + \tau V)\frac{S^*}{S} + (\mu + \omega)E + \frac{\beta_1 SIE^*}{E(m_1 S + m_2 I)} \\
&+ \frac{\beta_2 SBE^*}{E(K + B)} + I^*(\mu + p + \alpha + \frac{\omega E}{I}) + (\delta + \mu)R + \frac{\alpha I R^*}{R}(\tau + \mu)V\frac{\gamma SV^*}{V}
\end{aligned}
$$

If $P < M$, then $\frac{dL}{dt} < 0$ and $\frac{dL}{dt} = 0$ if and only if $S = S^*$, $E = E^*$, $I = I^*$, $R = R^*$, $V = V^*$ and $B = B^*$. The singleton of E_1 is the largest compact invariant set in $(S^*, E^*, I^*, R^*, V^*, B^*) \in \mathbb{R}^6{}_+ : \frac{dL}{dt} = 0$. Based on ([1,14]), this indicates that E_1 is globally asymptotically stable in $\mathbb{R}^6{}_+$ if $P < M$ according to LaSalle's invariant principle. □

3.11. Analysis of Sensitivity

The goal of the basic reproduction number sensitivity analysis inquiry is to determine which parameter value (Table 1) is more significant for anthrax disease spread. I compute the relative sensitivity of R_0 using Martcheva [20] in order to obtain the answer via the sensitivity index analysis.

Table 1. Description of model parameters.

Parameters	**Details of the Parameters**
λ	Recruitment rate
β_1	Host to host transmission coefficient (ratio dependent form)
m_1, m_2	Half saturation constant for ratio dependent host transmission
β_2	Env't transmission coefficient (Holling II in B)
k	Half saturation constant for env't transmission
ω	Rate of exposed to infectious (progression)
p	Production rate of spores by infections (terminal) animals
δ	Decay(clearance) rate of spores in env't
μ	Natural death rate
γ	Vaccination rate
α	Animal recovery rate
σ	Waning recovery rate
τ	Waning immunity of vaccinated animals

$$
\begin{aligned}
\Gamma_{\beta_1}^{R_0} &= \frac{\beta_1 k\delta(\gamma\mu)}{\beta_1 k\delta(\mu+\gamma)+\beta_2 p\lambda m_1}.\\
\Gamma_{\beta_2}^{R_0} &= \frac{\beta_2 p\lambda}{\beta_1 k\delta(\mu+\gamma)+m_1\beta_2 p\lambda}.\\
\Gamma_{\mu}^{R_0} &= -\mu\frac{\omega\beta_1(2\mu+p+\alpha)k\delta(\mu+\gamma)^2+\beta_2 m_1\omega p\lambda Q}{(\mu+p+\alpha)(\mu+\omega)k\delta(\mu+\gamma)\omega(\beta_1 k\delta(\mu+\gamma)+\beta_2 p\lambda m_1)}\\
\Gamma_{p}^{R_0} &= p\frac{\beta_2\lambda m_1(\mu+\alpha)-\beta_1 k\delta(\mu+\gamma)}{(\mu+p+\alpha)((\beta_1\delta k(\mu+\gamma)+m_1\beta_2 p\lambda))}\\
\Gamma_{\alpha}^{R_0} &= -\frac{\beta_1 k\delta\alpha}{m_1(\mu+p+\alpha)(\beta_1 k\delta(\mu+\gamma)+\beta_2 p\lambda m_1)}\\
\Gamma_{k}^{R_0} &= -\frac{\beta_2 p\lambda m_1}{\beta_1 k\delta(\mu+\gamma)+\beta_2 p\lambda m_1}\\
\Gamma_{m_1}^{R_0} &= -\frac{\beta_1 k\delta(\mu+\gamma)m_1}{\beta_1 k\delta(\mu+\gamma)+\beta_2 p m_1\lambda}\\
\Gamma_{\gamma}^{R_0} &= -\frac{\gamma\beta_2 p\lambda m_1 k\delta}{\beta_1 k\delta(\mu+\gamma)+\beta_2 p m_1\lambda}
\end{aligned}
$$

where

$$
Q = \frac{a_1(\mu+\gamma)+a_2(\mu+\gamma)+a_2a_3}{(a_2a_1(\mu+\gamma))^2}
$$

The R_0 sensitivity indices for the parameters listed in Table 2.

Table 2. Sensitivity index values in numbers for R_0.

Parameters	Sensitivity Indices	Values
β_1	+ve	+0.0054677
β_2	+ve	+0.36706
p	+ve	+0.1327
m_1	−ve	−0.0022694
k	−ve	−0.47907
α	−ve	−0.037231
γ	−ve	−0.39564
μ	−ve	−0.014919

4. Simulation and Discussions

In this section, MATLAB software is used to perform numerical simulations illustrating the dynamic behavior of the model. The initial conditions are taken as $S(0) = 59{,}000, E(0) = 30{,}000, I(0) = 15{,}000, R(0) = 3000, V(0) = 9000$, and $B(0) = 500$ per 100,000 animals as the starting conditions for the numerical simulation, in addition to the values of the parameters displayed in Table 3.

Table 3. Parameter values in the model.

Parameters	Values	Source
λ	1000	Assumed
β_1	0.1	[4]
β_2	0.2	Assumed
m_1	1	[4]
m_2	1	[4]
γ	0.1	[4]
τ	0.03	[9]
σ	0.2	[9]
α	0.01	[9]
ω	0.5	[21]
p	0.1	[22]
δ	0.1	[22]
k	1	Assumed
μ	0.01	[6]

Numerical simulations of system (1) were performed, and the resulting graphs are presented and interpreted as follows:

In Figure 2a with $R_0 = 0.016$, I observe that for the basic reproduction number $R_0 < 1$, all solution curves go to the anthrax-free equilibrium point. As a result, the anthrax goes extinct, and eradicated. In Figure 3, when $R_0 < 1$ and taking different initial conditions for the state variables show, all trajectories of state variables go to their components of the anthrax-free equilibrium point. These indicate that the anthrax free equilibrium point is globally asymptotically stable for the values of $R_0 < 1$. This implies that whatever anthrax persists in the population in the long run, the disease will be eradicated from the population.

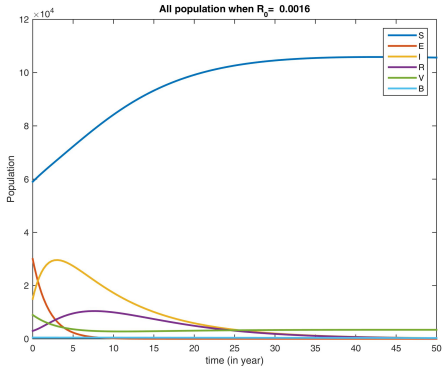
In Figure 2b with R_0 = 12.659, I observe that for the basic reproduction number $R_0 > 1$, all solution curves go away from the anthrax-free equilibrium point. These indicate that the anthrax-free equilibrium point is unstable for values of $R_0 > 1$, and the solutions will go to the endemic equilibrium point. Consequently, the anthrax invades a population. In Figure 4 with $R_0 > 1$ and different initial conditions for the state variables, all trajectories of state variables go to their components of the endemic equilibrium point. These indicate that the endemic equilibrium point is globally asymptotically stable for the values of $R_0 > 1$.

Figure 5 and Table 2 show that the sensitivity indexes of the basic reproduction number with regard to the model parameters. These parameters, which positive indices (β_1, β_2 and p) show that they have a great impact on the expansion of the anthrax disease in the community if their values increase. The reason that the basic reproduction number increases is because their values increase, which means that the average number of secondary cases of anthrax disease increases in the community. And also the parameters in which their sensitivity indices are negatives (m_1, α, γ and μ) have an influence on reducing at least the burden of the disease in the population; their values increase while the others are left constant. And also, as their values increase, the number of basic reproductions decreases, which leads to minimizing the endemicity of the disease in the community. Therefore, with sensitivity analysis, one can gain insight into the appropriate intervention strategies to prevent and control the spread and behavior of the disease described by model (1).

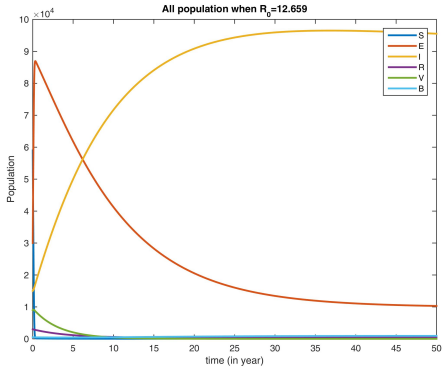
In Figure 6, I observe that as the environmental transmission coefficient rate increases, the more animals join the infected compartment from the exposed class. The exposed population decreases significantly over time and infected population increases. This is due to the fact the environmental transmission coefficient rate with infected individuals increases, then the reproduction number increases, which means the infection persists in the population. And it is realized that the population of infected animals will rise as the environmental transmission coefficient rate increases. Hence, to decrease the number of infected animals, one can decrease the environmental transmission coefficient rate.

Figure 7 shows that as the waning immunity of vaccinated animals rate grows, the vaccinated population grows while susceptible population decreases. Because if the waning immunity of vaccinated animals rate of susceptible animals increases, then the reproduction number decreases.

In Figure 8, I observe that increasing the vaccination rate minimizes the number of infected animals while the number of susceptible animals increases. This is because when the vaccination rate of susceptible animals rises, the number in the infected compartment declines. Further, this graph demonstrates that the vaccination rate a large impact on the rate of susceptible to exposed and exposed to infectious. Hence, to decrease the number of infected animals, one can increase the rate of vaccination of susceptible animals.

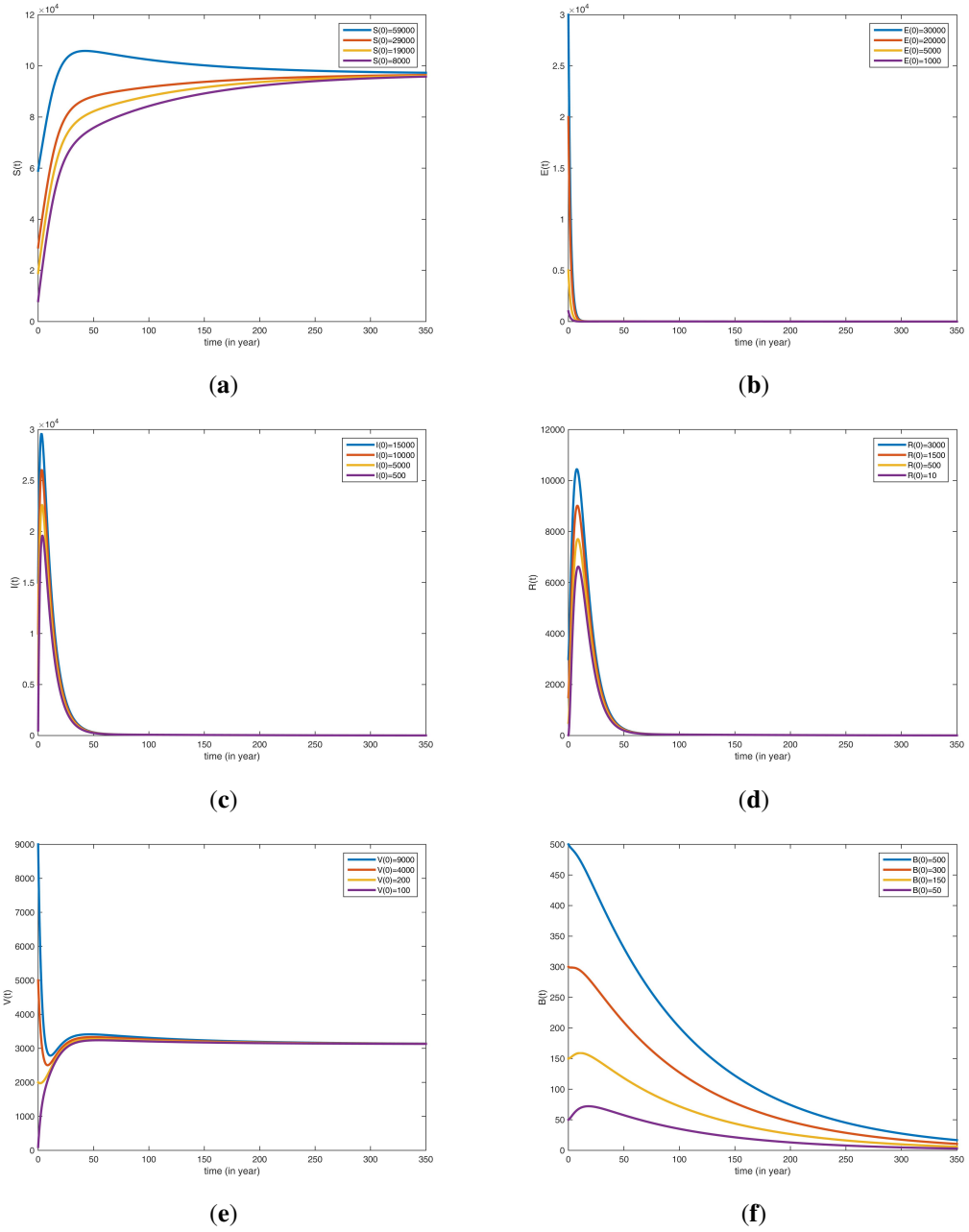


(a) Trajectories of State variable for R_0 = 0.016.



(b) Trajectories of State variable R_0 = 12.659.

Figure 2. Trajectories of state for different R_0 value.

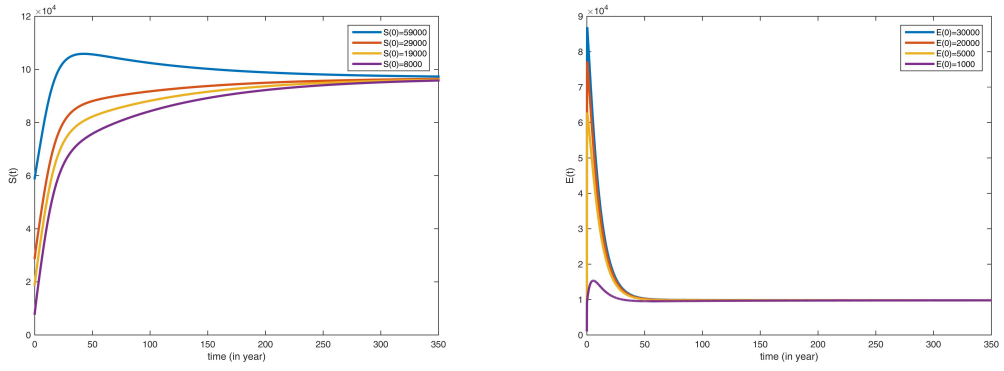


(**a**) (**b**)

(**c**) (**d**)

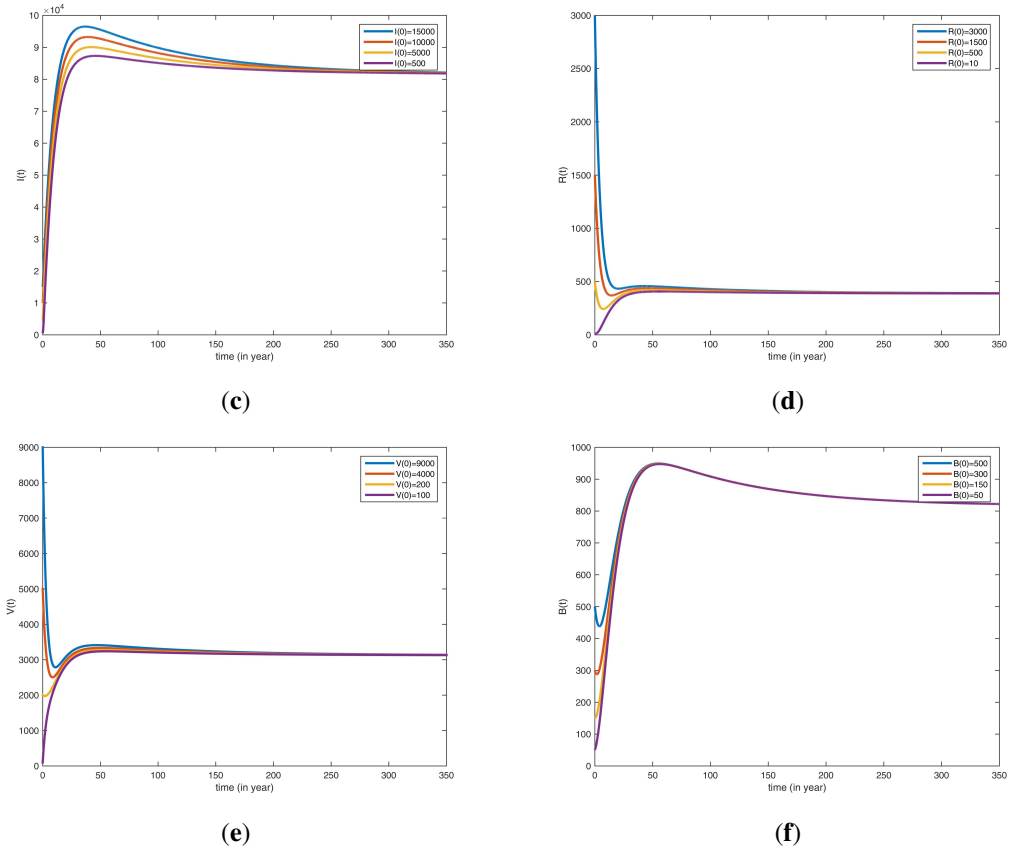
(**e**) (**f**)

Figure 3. Trajectories of state variables for $R_0 = 0.016$ with different initial conditions.



(**a**) (**b**)

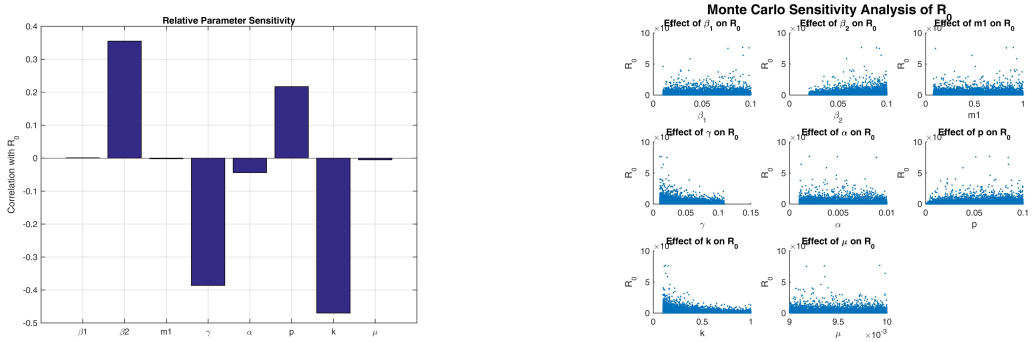
Figure 4. *Cont.*



(**c**) (**d**)

(**e**) (**f**)

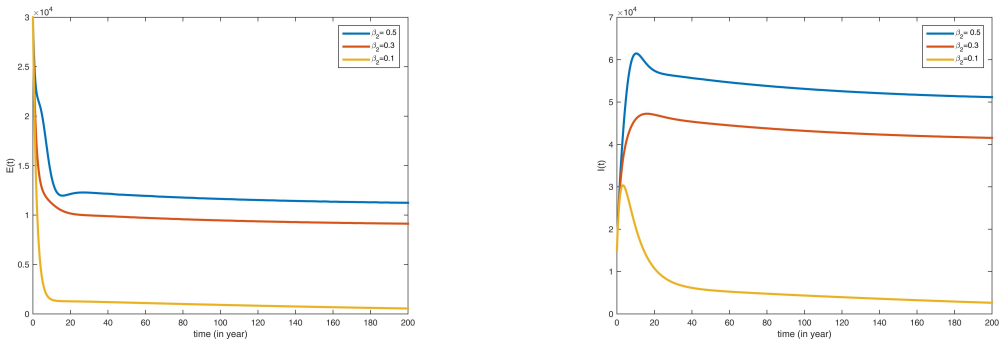
Figure 4. Trajectories of state variables for R_0 = 12.659 with different initial conditions.



(**a**) Sensitivity indices that measure each parameter's impact on R_0

(**b**) Scatter graphs showing how (R_0) varies and is related to different parameter ranges

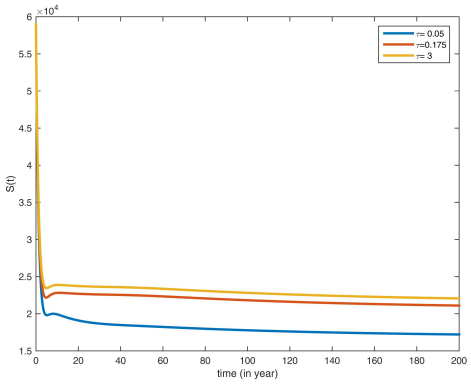
Figure 5. Sensitivity analysis of the basic reproduction number (R_0) relative to model parameters.



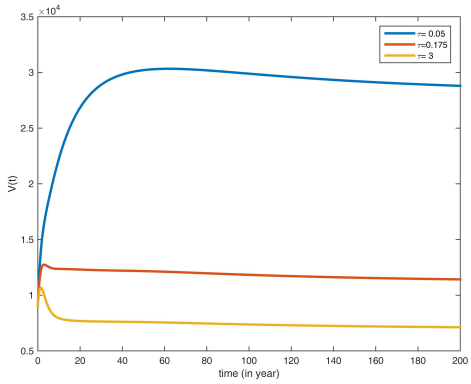
(**a**) Exposed population with varying environmental transmission coefficient rate (β_2)

(**b**) Infected population with varying environmental transmission coefficient rate (β_2)

Figure 6. Exposed and infected population with varying environmental transmission coefficient rate (β_2).

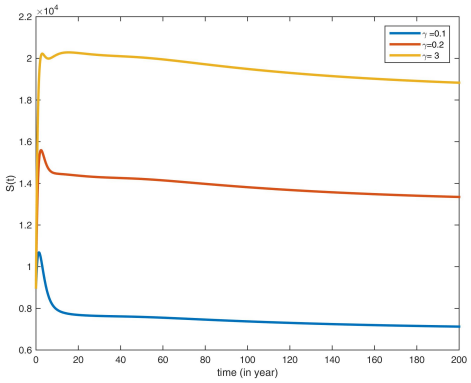


(**a**) Susceptible population with varying waning immunity of vaccinated animals rate (τ)

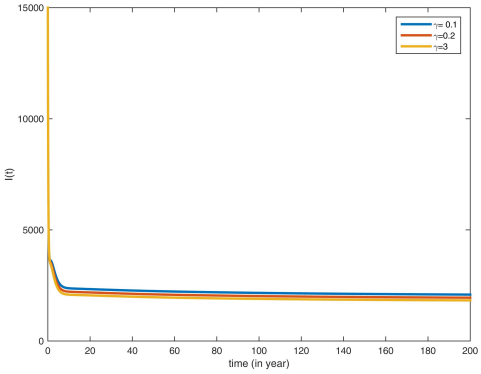


(**b**) Vaccinated population with varying waning immunity of vaccinated animals rate (τ)

Figure 7. Susceptible and vaccinated population with varying waning immunity of vaccinated animals rate (τ).



(**a**) Susceptible population with different rates of vaccination rate (γ)



(**b**) Infected population with different rates of vaccination (γ)

Figure 8. Susceptible and Infected population with different rates of vaccination (γ).

5. Conclusions

In order to examine the dynamics of anthrax disease transmission, I developed a mathematical model in this study. By incorporating essential compartments and epidemiological elements, this model provides a comprehensive framework for understanding disease trends. I calculated the basic reproduction number (R_0), which serves as the key criterion for my analysis. According to my findings, the anthrax disease-free equilibrium is stable both locally and globally when $R_0 < 1$, indicating the existence of effective control mechanisms. However, when $R_0 > 1$, the endemic equilibrium stabilizes, suggesting the urgent need for intervention actions.

I recommend policymakers and stakeholders to give great attention in minimizing the effect of the environmental transmission coefficient rate as it have high impact on increasing the infected number of animals. Also provide appropriate vaccines at right time for susceptible animals as I observed increasing the vaccination rate decreases the infected number of animals.

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Institutional Review Board Statement

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Informed Consent Statement

Not applicable.

Data Availability Statement

The data used to support this model is sourced from previously published articles, which are appropriately referenced in this study. However, some of the assumptions made are suitable for the proposed simulation.

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Conflicts of Interest

The author declares no conflict of interest.

Use of AI and AI-Assisted Technologies

During the preparation of this work, the author used ChatGPT (OpenAI) to assist with language editing and improving the clarity of the manuscript. After using this tool, the author reviewed and edited the content as needed and takes full responsibility for the content of the published article.

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