

Optimal Control Analysis of Anthrax Dynamics in Human and Animal Populations

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Abstract

Anthrax, caused by *Bacillus anthracis*, is a severe zoonotic disease with substantial health and economic consequences for animal and human populations. This study developed a nonlinear mathematical model to examine anthrax transmission incorporating animal vaccination and extended the model to identify optimal control strategies. The analysis examined equilibrium stability, parameter sensitivity, and intervention strategies. The disease-free equilibrium was stable when the basic reproduction number was below one, whereas the endemic equilibrium was stable when it exceeded one. Sensitivity analysis indicated that reducing cattle infection rates and the probability of human infection, alongside improvements in control-related parameters, could reduce disease levels. Optimal control analysis identified early human intervention, particularly prompt antibiotic treatment (u_2), as essential for achieving elimination within the model. Combining prevention with early treatment was the most cost-effective strategy, while strategies excluding treatment could worsen outbreaks. Although combinations of multiple controls could achieve elimination, they yielded diminishing returns, with the u_1 – u_2 combination identified as the most efficient approach. These findings emphasise the importance of integrating prevention with prompt treatment and provide a mathematical basis for prioritising efficient anthrax control strategies.

Keywords: Anthrax Modelling; Animal Vaccination; Stability Analysis; Sensitivity Analysis; Optimal Control

INTRODUCTION.

Anthrax, derived from a Greek word meaning “coal-like,” refers to the black skin lesion it causes. It is a global zoonotic disease caused by *Bacillus anthracis*, mainly affecting herbivores but transmissible to humans through infected animals or contaminated products. Notably, it does not spread from human to human.

Mathematical models are essential for understanding and controlling infectious diseases, offering insights into transmission dynamics and aiding prediction and prevention (Osman, *et al.*, 2018). According to Githire *et al.*, (2012), anthrax in animals can be effectively controlled through vaccination and treatment, as immunity is temporary and symptoms may appear after a short incubation period.

The deterministic model for anthrax transmission dynamics in both human and animal populations, incorporating vaccination for animals was developed (Osman *et al.* 2020). Their qualitative analysis determined the basic reproduction number (R_{hv}), equilibrium points, and performs sensitivity analysis to identify key parameters influencing disease spread. The analysis reveals that reducing human–animal interaction rates decrease R_{hv} , while the disease-free equilibrium is locally asymptotically stable when $R_{hv} < 1$. The study extends to optimal control, comparing multiple intervention strategies, and finds through infection averted ratio (IAR) analysis that the most effective and cost-efficient approach is the combination of animal vaccination and human prevention efforts. The findings emphasize that public education on vaccination and the risks associated with consuming infected animal products should be prioritized to effectively combat anthrax outbreaks.

Akande *et al.*, (2024) introduced a novel risk-structured model for anthrax, partitioning the human population into high-risk and low-risk susceptible classes to capture the effect of behavioral changes and education. Their comprehensive analysis included bifurcation analysis, which demonstrated a forward bifurcation at $R_c = 1$, and a rigorous global stability analysis using Lyapunov functions. A key contribution is the detailed sensitivity analysis, which identified the most critical parameters for disease control, such as the effective transmission rate in animals (β_1), and highlighted that mitigation measures targeting these parameters would be most effective in reducing the effective reproduction number R_c .

A mathematical model exploring anthrax transmission dynamics using a saturated incidence rate, incorporating vaccination compartments for both human and animal

populations simultaneously, was developed by (Michael, *et al.*, 2024). Their qualitative and quantitative analyses, including local and global stability tests, showed the model to be locally asymptotically stable when the basic reproduction number is less than one, with no backward bifurcation present. Numerical simulations from their study indicated that increasing vaccination rates in both populations effectively reduces the spread of anthrax, supporting vaccination as a primary control measure.

A public health perspective on the recent 2023 anthrax outbreaks in Nigeria, linking them to the importation of contaminated animal products by nomadic pastoralists crossing national borders, was provided by (Mogaji, *et al.*, 2024). The review emphasizes the urgent need for improved surveillance, molecular diagnostics to understand the genetic diversity of circulating strains, and interdisciplinary action under a One Health framework. This paper highlights the critical real-world drivers of anthrax transmission such as livestock movement, trade, and community practices that mathematical models must account for to be realistic and policy-relevant.

Raza and Abdella (2024) investigated the nonlinear dynamics of an anthrax transmission model with a time delay, specifically incorporating the incubation period into the infection term. Their analytical work involved deriving the reproduction number and establishing conditions for the local and global stability of both disease-free and endemic equilibria. The numerical simulations confirmed that the delay mechanism plays a crucial role; by implementing effective control strategies during this delay period, the disease dynamics can be significantly slowed, and the infected population can be reduced to zero, demonstrating the potential of delay-based interventions.

Triska and Zevika (2025) focused on a mathematical model (SVICA) for anthrax transmission in herbivorous animals, explicitly incorporating the roles of vaccination and harvesting. Their model included compartments for carcasses and spores, recognizing the environment as a key reservoir. By calculating the basic reproduction number R_0 and analyzing bifurcations, they discovered that for certain parameter values when $R_0 < 1$, the endemic equilibrium could become unstable, leading to limit cycles. This finding revealed two distinct oscillatory behaviors, including a slow-fast limit cycle that explains periods of low disease incidence followed by sudden outbreaks, highlighting the complex periodic nature of anthrax dynamics.

Despite increasing model sophistication, most existing anthrax transmission frameworks still reduce interventions to a single intermediate stage and seldom incorporate psychologically driven protective behavior alongside distinct infection pathways within a unified mathematical structure. This study addresses these gaps by extending the framework of Osman, Otoo, and Makinde. We propose an optimal control model in which the infected human class is subdivided into early-detected and late-detected cases. Within an optimal control setting, individual susceptibility is dynamically adjusted, with risk perception informing time-dependent self-protective behavior. Concurrently, multiple curative interventions across both human and cattle populations are modeled as flexible, time-varying controls. By integrating preventive and curative strategies as adaptive human and cattle responses within a single quantitative framework, the model clarifies the synergistic effects of preventive behavior, early treatment of detected cases, and management of late-stage infections (e.g., hospitalization). It further highlights the critical role of vaccination efficacy in susceptible cattle in driving the system toward the epidemiological threshold necessary for anthrax elimination.

1. Model Formulation and Description

The model divides human and animal populations at any time, (t) into compartments with respect to their disease status as shown in figure 1. Total human population, $N_h(t)$, is subdivided into susceptible human (S_h), early detected infected human (I_{ED}), late detected infected human (I_{LD}), and recovered human (R_h).

Hence total human population;

$$N_h(t) = S_h(t) + I_{ED}(t) + I_{LD}(t) + R_h(t) \quad (1)$$

Total animal population denoted by $N_c(t)$, is subdivided into vaccinated animal (V_c), susceptible animal (S_c), infected animal (I_c) and recovered animal (R_c).

Hence total animal population;

$$N_c(t) = V_c(t) + S_c(t) + I_c(t) + R_c(t) \quad (2)$$

Susceptible humans are recruited at a rate θ_h . Human acquire anthrax through direct contact, inhalation and ingestion of contaminated animal products at a rate $(I_c + I_{ED} + I_{LD})\beta$. Late detected infected humans recover from anthrax disease at a rate τ_h and early detected

infected humans recover at a rate γ_h while humans infected with anthrax die at a rate δ_h . The entire human population has a natural death rate of μ_h .

Susceptible animals are recruited at a rate θ_c . The disease can be obtained through contacts with infectious animals and humans at a rate $(I_c + I_{ED} + I_{LD})\beta$. Natural death rate of all animal compartments is μ_c and death rate as a result of anthrax infection is δ_c . Animal recovery rate is τ_c . The system of ordinary differential equations obtained from the model in figure 1 are as follows:

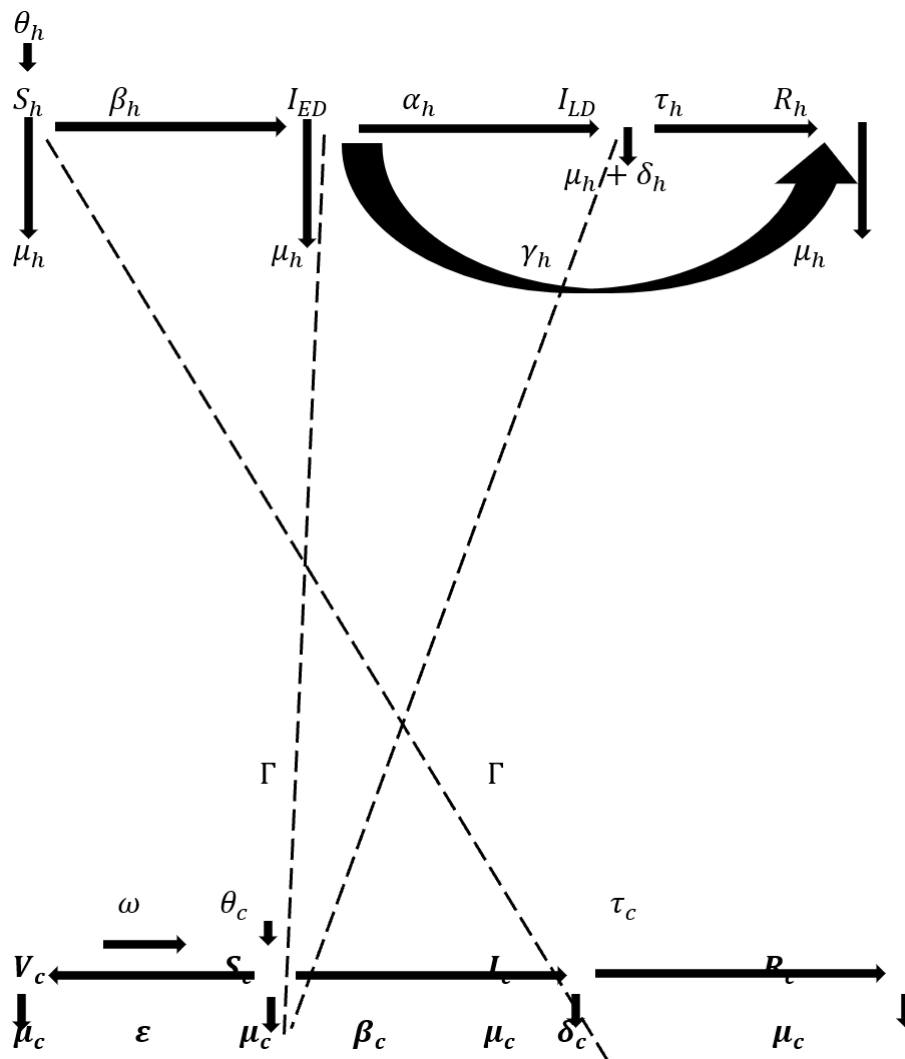


Figure 1. Flow diagram for the anthrax disease transmission dynamics

The model equations are:

$$\frac{dS_h}{dt} = \theta_h - \beta_h \Gamma S_h - \mu_h S_h$$

$$\begin{aligned}
 \frac{dI_{ED}}{dt} &= \beta_h \Gamma S_h - (\mu_h + \alpha_h + \gamma_h) I_{ED} \\
 \frac{dI_{LD}}{dt} &= \alpha_h I_{ED} - (\mu_h + \delta_h + \tau_h) I_{LD} \\
 \frac{dR_h}{dt} &= \tau_h I_{LD} + \gamma_h I_{ED} - \mu_h R_h \\
 \frac{dV_c}{dt} &= \theta_c \varepsilon - (\mu_c + \omega) V_c \\
 \frac{dS_c}{dt} &= \theta_c (1 - \varepsilon) + \omega V_c - \beta_c \Gamma S_c - (\mu_c + \varepsilon) S_c \\
 \frac{dI_c}{dt} &= \beta_c \Gamma S_c - (\mu_c + \tau_c) I_c \\
 \frac{dR_c}{dt} &= \tau_c I_c - \mu_c R_c
 \end{aligned} \tag{3}$$

Where $\Gamma = (1 - \phi)[I_c + I_{ED} + I_{LD}]$

Model Analysis

Positivity and Boundedness of Solutions

In epidemiological models, conditions under which the system should have non-negative solutions is paramount. The model would be biologically meaningful if all the solutions with positive initial conditions remain positive at every point in time.

$$\pi = \{(S_h(t), I_{ED}(t), I_{LD}(t), R_h(t), V_c(t), S_c(t), I_c(t), R_c(t)) \in \mathbb{R}^8:$$

Theorem 1. Let

$$(S_h(0), I_{ED}(0), I_{LD}(0), R_h(0), V_c(0), S_c(0), I_c(0), R_c(0)) > 0\}$$

Then the solution of $\{(S_h(t), I_{ED}(t), I_{LD}(t), R_h(t), V_c(t), S_c(t), I_c(t), R_c(t))\}$ are non-negative for all time $t \geq 0$.

Hence, if $(S_h(0), I_{ED}(0), I_{LD}(0), R_h(0), V_c(0), S_c(0), I_c(0), R_c(0))$, then $(S_h(t), I_{ED}(t), I_{LD}(t), R_h(t), V_c(t), S_c(t), I_c(t), R_c(t))$ are also non-negative for all $t > 0$.

Human total population at any given time:

$$N_h(t) = S_h(t) + I_{ED}(t) + I_{LD}(t) + R_h(t) \tag{4}$$

$$\frac{dN_h}{dt}(t) = \frac{dS_h}{dt} + \frac{dI_{ED}}{dt} + \frac{dI_{LD}}{dt} + \frac{dR_h}{dt} \tag{5}$$

$$= \theta_h - \mu_h N_h - \delta_h I_{LD}$$

In the absence of mortality due to anthrax infection, the above equation becomes:

$$\frac{dN_h}{dt} \leq \theta_h - \mu_h N_h \quad (6)$$

Solving the equation and as $t \rightarrow \infty$, the population size, $N_h \rightarrow \frac{\theta_h}{\mu_h}$

$$0 \leq N_h \leq \frac{\theta_h}{\mu_h} \text{ and } N_h(t) \leq \frac{\theta_h}{\mu_h}$$

Also if

$$N_h(0) \leq \frac{\theta_h}{\mu_h}, \text{ then } N_h(t) \leq \frac{\theta_h}{\mu_h}$$

$$\pi_h = \{(S_h, I_{ED}, I_{LD}, R_h) \in \mathbb{R}_+^4 : S_h + I_{ED} + I_{LD} + R_h \leq \frac{\theta_h}{\mu_h}\} \quad (7)$$

However, for total animal population at any given time:

$$N_c(t) = V_c(t) + S_c(t) + I_c(t) + R_c(t) \quad (8)$$

$$\frac{dN_c}{dt}(t) = \frac{dV_c}{dt} + \frac{dS_c}{dt} + \frac{dI_c}{dt} + \frac{dR_c}{dt} \quad (9)$$

$$= \theta_c - \mu_c N_c - \delta_c I_c$$

$$\frac{dN_c}{dt} \leq \theta_c - \mu_c N_c \quad (10)$$

Solving the differential equation and as $t \rightarrow \infty$, the population size, $N_c \rightarrow \frac{\theta_c}{\mu_c}$

$$0 \leq N_c \leq \frac{\theta_c}{\mu_c} \text{ and } N_c(t) \leq \frac{\theta_c}{\mu_c}$$

Also if,

$$N_c(0) \leq \frac{\theta_c}{\mu_c}, \text{ then } N_c(t) \leq \frac{\theta_c}{\mu_c} \quad (11)$$

$$\pi_c = \{(V_c, S_c, I_c, R_c) \in \mathbb{R}_+^4 : V_c + S_c + I_c + R_c \leq \frac{\theta_c}{\mu_c}\}$$

Hence, feasible region for the dynamical system in (1) is given by

$$\pi = \pi_h \times \pi_c \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4. \quad (12)$$

$$\pi_h = \{(S_h, I_{ED}, I_{LD}, R_h) \in \mathbb{R}_+^4 : S_h + I_{ED} + I_{LD} + R_h \leq \frac{\theta_h}{\mu_h}\} \quad (13)$$

$$\pi_c = \{(V_c, S_c, I_c, R_c) \in \mathbb{R}_+^4 : V_c + S_c + I_c + R_c \leq \frac{\theta_c}{\mu_c}\} \quad (14)$$

where π is positively invariant.

Disease Free Equilibrium

The disease free equilibrium (DFE) of the dynamical system in (3) only exists in the absence of infections. We can then compute DFE by setting the system of equations in (3) to zero and establish DFE points.

At DFE, there are no infections and recovery. Hence, $I_{ED}^*, I_{LD}^*, R_h^*, R_c^* = 0$

$$\xi_0 = (S_h^*, I_{ED}^*, I_{LD}^*, R_h^*, V_c^*, S_c^*, I_c^*, R_c^*)$$

$$\frac{dS_h}{dt} = \theta_h - \beta_h \Gamma S_h - \mu_h S_h = 0$$

$$\frac{dV_c}{dt} = \theta_c \varepsilon - (\mu_c + \omega) V_c = 0 \quad (15)$$

$$\frac{dS_c}{dt} = \theta_c (1 - \varepsilon) + \omega V_c - \beta_c \Gamma S_c - \mu_c S_c = 0$$

The DFE is given by ;

$$\xi_0 = \left[\frac{\theta_h}{\mu_h}, 0, 0, 0, \frac{\theta_c \varepsilon}{\mu_c + \omega}, \frac{\theta_c (1 - \varepsilon)(\mu_c + \omega) + \omega \theta_c \varepsilon}{\mu_c (\mu_c + \omega)}, 0, 0 \right] \quad (16)$$

The Basic Reproduction Number.

Using the next generation matrix approach, Muia, *et al.*, (2018) the reproductive rate can be established. The reproductive rate combines the biology of infections with social and behaviour of the factors influencing contact rate. The basic reproductive rate refers to the number of secondary cases one infectious individual will produce in a completely susceptible population (Okosun, *et al.*, 2016; Van den Driessche and Watmough, J. 2002). This is a threshold parameter that governs the spread of disease.

Definition 1. 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 $\rho(A)$ denotes the set of eigenvalues of the matrix A.

Considering only the infective compartments in the system of differential equations in (3):

$$\frac{dI_{ED}}{dt} = \beta_h \Gamma S_h - (\mu_h + \alpha_h + \gamma_h) I_{ED}$$

$$\frac{dI_{LD}}{dt} = \alpha_h I_{ED} - (\mu_h + \delta_h + \tau_h) I_{LD} \quad (17)$$

$$\frac{dI_c}{dt} = \beta_c \Gamma S_c - (\mu_c + \delta_c + \tau_c) I_c$$

Let f be the number of new infection coming into the system and v be the number of infectives that are leaving the system either by death or birth.

$$F_i = \begin{bmatrix} \beta_h \psi (I_C + I_{ED} + I_{LD}) S_h \\ 0 \\ \beta_c \psi (I_C + I_{ED} + I_{LD}) S_c \end{bmatrix}$$

Where $\psi = (1 - \phi)$

$$V_i = \begin{bmatrix} K_1 I_{ED} \\ -\alpha_h I_{ED} + K_2 I_{LD} \\ K_4 I_C \end{bmatrix}$$

The jacobian matrix of f and v at DFE is obtained by f and v as follows:

$$F = \begin{bmatrix} \frac{\beta_h \psi \theta_h}{\mu_h} & \frac{\beta_h \psi \theta_h}{\mu_h} & \frac{\beta_h \psi \theta_h}{\mu_h} \\ 0 & 0 & 0 \\ \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3} & \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3} & \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3} \end{bmatrix}$$

$$V = \begin{bmatrix} K_1 & 0 & 0 \\ -\alpha_h & K_2 & 0 \\ 0 & 0 & K_4 \end{bmatrix}$$

Computing the product of F and V^{-1}

$$= \begin{bmatrix} \frac{\beta_h \psi \theta_h}{\mu_h K_1} + \frac{\beta_h \psi \theta_h \alpha_h}{\mu_h K_1 K_2} & -\frac{\beta_h \psi \theta_h}{\mu_h K_2} & \frac{\beta_h \psi \theta_h}{\mu_h K_4} \\ 0 & 0 & 0 \\ \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_1 K_3} + \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon) \alpha_h}{\mu_c K_1 K_2 K_3} & -\frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_2 K_3} & \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3 K_4} \end{bmatrix}$$

$F V^{-1}$ will give rise to a polynomial in λ of order n , this is called the Characteristic Polynomial, which when set to zero and solved, gives rise to the eigen values $\lambda_1 \dots \lambda_n$ i.e.

$|F V^{-1} - \lambda| = 0$ which is characteristic equation.

$$\begin{bmatrix} \lambda - \frac{\beta_h \psi \theta_h}{\mu_h K_1} - \frac{\beta_h \psi \theta_h \alpha_h}{\mu_h K_1 K_2} & \frac{\beta_h \psi \theta_h}{\mu_h K_2} & \frac{\beta_h \psi \theta_h}{\mu_h K_4} \\ 0 & \lambda & 0 \\ -\frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_1 K_3} - \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon) \alpha_h}{\mu_c K_1 K_2 K_3} & \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_2 K_3} & \lambda - \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3 K_4} \end{bmatrix} = 0$$

By computing the eigenvalues of FV^{-1} and selecting the dominant eigenvalues,

$$R_0^h = \frac{\beta_h \psi \theta_h}{\mu_h K_1} + \frac{\beta_h \psi \theta_h \alpha_h}{\mu_h K_1 K_2} = \frac{\beta_h \psi \theta_h (K_2 + \alpha_h)}{\mu_h K_1 K_2} \quad (18)$$

and

$$R_0^c = \frac{\beta_c \psi \theta_c (K_3 + \varepsilon \omega - K_3 \varepsilon)}{\mu_c K_3 K_4} \quad (19)$$

Global Stability of the Disease Free Equilibrium

Proposition 3. The disease free equilibrium (DFE) of model (3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 4. The disease free equilibrium given by E^0 , the model (3) is globally asymptotically stable if $R_0 < 1$.

Proof.

Using the Castillo Chavez theorem (Chavez *et al.*, 2002). Let $X(t)$ and $Y(t)$ represent the compartments defined by the uninfected and infected classes of the system (3), respectively.

Thus, system (3) can be written as

$$\frac{dX}{dt} = F(X, Y), \frac{dY}{dt} = G(X, Y); \quad G(X, 0) = 0. \quad (20)$$

Where F and G are the corresponding right-hand side of the system. According to Castillo Chavez theorem, to establish the global asymptotic stability of the anthrax-free equilibrium, the following two conditions (D1) and (D2) must be satisfied for $R_0 < 1$.

- (D1) $X^0 = (1, 0, 1, 1, 0)^T$ is globally asymptotically stable for $\frac{dX}{dt} = F(X, 0)$.
- (D2) $G \geq 0$ where $G(X, Y) = AY - G(X, Y)$ and $A = D_Y G(X^0, 0)$ is Metzler matrix $\forall (X, Y) \in \Omega$

The first Condition is globally asymptotically stability of X^0 , we have

$$\frac{dX}{dt} = F(X, 0) = \begin{pmatrix} \theta_h - \mu_h S_h \\ -\mu_h R_h \\ \theta_c \varepsilon - (\mu_c + \omega) V_c \\ \theta_c (1 - \varepsilon) - \omega V_c - \mu_c S_c \\ -\mu_c R_c \end{pmatrix} \quad (21)$$

by solving the above system, we have the behaviour of each compartment as follows

$$X^0 = \begin{pmatrix} S_h \\ R_h \\ V_c \\ S_c \\ R_c \end{pmatrix} = \begin{pmatrix} 1 + S_h(0)e^{-\mu_h t} \\ R_h(0)e^{-\mu_h t} \\ 1 + V_c(0)e^{-(\mu_c + \omega)t} \\ 1 + S_c(0)e^{-\mu_c t} \\ R_c(0)e^{-\mu_h t} \end{pmatrix} \quad (22)$$

Now since $\lim_{t \rightarrow \infty} X(t) = X^0$ satisfying the first condition. For the second condition (D2)

$$A = \begin{pmatrix} \beta_h \psi S_h - K_1 & \beta_h \psi S_h & \beta_h \psi S_h \\ \alpha_h & -K_2 & 0 \\ \beta_c \psi S_c & \beta_c \psi S_c & \beta_c \psi S_c - K_4 \end{pmatrix} \quad (23)$$

Then we compute

$$\hat{G}(X, Y) = AY - G(X, Y) = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \quad (24)$$

It is obvious that $\hat{G}(X, Y) = 0$, thus $\hat{G}(X, Y) \geq 0$ in (D2) is satisfied. Hence, E^0 is globally asymptotically stable, provided that $R_0 \leq 1$. This completes the proof.

Global Stability of Endemic Equilibrium

In this section, the global behaviour of the system in (3) is analyzed. Considering the non-linear Lyapunov function.

Theorem 5. The Anthrax -present equilibrium point E^* is globally asymptotically stable (GAS) if $R_0 > 1$.

The EE can only exist if and only if $R_0 > 1$. So by letting $R_0 > 1$, it implies that the EE exists.

Considering the non-linear Lyapunov function below;

$$L = c_1 S_h + c_2 I_{ED} + c_3 I_{LD} + c_4 R_h + c_5 V_c + c_6 S_c + c_7 I_C + c_8 R_c \quad (25)$$

Where c_i for $i = 1, 2, 3, \dots, 8$ are constants to be chosen during the proof are defined. The derivative of L along the solution of (1) is given by

$$\frac{dL}{dt} = c_1 \frac{dS_h}{dt} + c_2 \frac{dI_{ED}}{dt} + c_3 \frac{dI_{LD}}{dt} + c_4 \frac{dR_h}{dt} + c_5 \frac{dV_c}{dt} + c_6 \frac{dS_c}{dt} + c_7 \frac{dI_C}{dt} + c_8 \frac{dR_c}{dt} \quad (26)$$

gives

$$\begin{aligned} \frac{dL}{dt} = & c_1 (\theta_h - \beta_h \Gamma S_h - \mu_h S_h) + c_2 (\beta_h \Gamma S_h - (\mu_h + \alpha_h + \gamma_h) I_{ED}) + c_3 (\alpha_h I_{ED} - (\mu_h + \delta_h + \tau_h) I_{LD}) \\ & + c_4 (\tau_h I_{LD} + \gamma_h I_{ED} - \mu_h R_h) + c_5 (\theta_c \varepsilon - (\mu_c + \omega) V_c) + c_6 (\theta_c (1 - \varepsilon) + \omega V_c - \beta_c \Gamma S_c - \mu_c S_c) \\ & + c_7 (\beta_c \Gamma S_c - (\mu_c + \delta_c + \tau_c) I_C) + c_8 (\tau_c I_C - \mu_c R_c) \end{aligned} \quad (27)$$

$$\begin{aligned} \frac{dL}{dt} = & c_1 (\theta_h - \mu_h S_h) + (c_2 - c_1) \beta_h \Gamma S_h + (c_3 - c_2) \alpha_h I_{ED} - c_2 \mu_h I_{ED} + (c_4 - c_2) \gamma_h I_{ED} \\ & - c_3 (\mu_h + \delta_h) I_{LD} + (c_4 - c_3) \tau_h I_{LD} - c_4 \mu_h R_h - c_5 \mu_c V_c + (c_6 - c_5) (\theta_c \varepsilon + \omega V_c) \\ & + c_6 (\theta_c - \mu_c S_c) + (c_7 - c_6) \beta_c \Gamma S_c - c_7 (\mu_c + \delta_c) I_C + (c_8 - c_7) \tau_c I_C - c_8 \mu_c R_c \end{aligned} \quad (28)$$

Taking $c_1, c_2, c_3, c_4, c_5, c_6, c_7$ and c_8 such that $c_1 = c_2 = c_3 = c_4 = c_5 = c_6 = c_7 = c_8$, $\theta_h - \mu_h S_h = 0$ and $\theta_c - \mu_c S_c = 0$ gives

$$\frac{dL}{dt} = -c_2 \mu_h I_{ED} - c_3 (\mu_h + \delta_h) I_{LD} - c_4 \mu_h R_h - c_5 \mu_c V_c - c_7 (\mu_c + \delta_c) I_C - c_8 \mu_c R_c \quad (29)$$

Since L is positive definite and its time derivative, $\frac{dL}{dt}$, is negative definite, L acts as a valid

Lyapunov function for the system (3). According to Lyapunov's stability theorem (Hubbard J.H. *et al.* 2012), the endemic equilibrium E^* is globally asymptotically stable within the invariant region Ω .

Sensitivity Analysis

In this section, we have illustrated a brief of the sensitivity analysis of the basic reproduction number R_0 . This enables the determination of the parameters that will give a rigorous shift in the threshold ratio R_0 of the model, this means that a change in a sensitive

parameter will yield a great quantitative difference in R_0 and may bring about qualitative changes. Here, the index of forward sensitivity analysis called the elasticity index (Rodrigues H. S. *et al.* 2013) is applied and it is defined as the proportion between the absolute change of R_0 and the absolute change of the parameter corresponding to the relative change as the follow:

$$Y_{\ell}^{R_0} = \frac{\ell}{R_0} \times \frac{\partial R_0}{\partial \ell} \quad (30)$$

Sensitivity Indices of R_0

Since $R_{0H\&c}$, R_{0H} , R_{0c} are the basic reproduction number for both humans and Anthrax respectively. The sensitivity analysis of R_0 to each of its parameters will be evaluated via the sensitivities of each $R_{0H\&c}$, R_{0H} , R_{0c} such that

$$Y_{\ell_1}^{R_{0H\&c}} = \frac{\ell_1}{R_{0H\&c}} \times \frac{\partial R_{0H\&c}}{\partial \ell_1}, \quad Y_{\ell_2}^{R_{0H}} = \frac{\ell_2}{R_{0H}} \times \frac{\partial R_{0H}}{\partial \ell_2}, \quad Y_{\ell_3}^{R_{0c}} = \frac{\ell_3}{R_{0c}} \times \frac{\partial R_{0c}}{\partial \ell_3} \quad (31)$$

where ℓ_1 , ℓ_2 , ℓ_3 denotes the parameter related to $R_{0H\&c}$, R_{0H} , and R_{0c} , respectively. Via the obvious expression of the basic reproduction number R_0 per the baseline values of parameters enumerated in Table 1, the values of the sensitivities indices are shown in Table 2.

Table 1: Lists of parameters and variables that were employed along with their daily values.

Variables	Descriptions	Variables	Descriptions
S_h	Susceptible human	V_c	Vaccinated cattle
I_{ED}	Early detected infected human	S_c	Susceptible cattle
I_{LD}	Late detected infected human	I_c	Infected cattle
R_h	Recovered human	R_c	Recovered cattle

Symbols	Description	Value
θ_h	Recruitment rate into the human population	0.92
Γ	Force of infection	
μ_h	Natural death rate for humans	0.012
ϕ	Public awareness campaign	0.45

α_h	Progression from early infection to late infection	0.15
τ_h	Recovery rate for late detected class	0.04
γ_h	Recovery rate for early detected class	0.25
δ_h	Anthrax induced death rate for humans	0.5
β_h	Probability of infection for humans	0.01
θ_c	Recruitment rate into the cattle population	0.99
μ_c	Natural death rate for cattles	0.0001
ω	Loss of immunity of vaccinated cattles	0.02
ε	Rate of successful vaccination	0.70
δ_c	Anthrax induced death rate for cattles	0.45
τ_c	Recovery rate for cattles	0.025
β_c	Probability of infections for cattles	0.05

Table 2: Sensitivity indices of R_{0H} and R_{0C}

Parameter	Sign	$\Upsilon_{\ell_1}^{R_{0H\&c}}$	Parameter	Sign	$\Upsilon_{\ell_2}^{R_{0H}}$	Parameter	Sign	$\Upsilon_{\ell_3}^{R_{0c}}$
ϕ	-ve	1.0046×10^{-4}	ϕ	-ve	0.8181	ϕ	-ve	1.0046×10^{-4}
ε	-ve	0.4705	α_h	-tve	0.0795	ε	-ve	0.4705
α_h	-tve	7.9313×10^{-6}	δ_h	-tve	0.3004	δ_c	-tve	0.5925
δ_c	-tve	0.5925	γ_h	-ve	0.4166	μ_c	-tve	1.0369
δ_h	-tve	4.6564×10^{-6}	μ_h	-tve	1.3800	τ_c	-tve	0.3703
γ_h	-ve	4.1545×10^{-5}	τ_h	-tve	0.0770	θ_c	+tve	1.0000
μ_c	-tve	1.0369	θ_h	+tve	1.0000	β_c	+tve	1.0000
μ_h	-tve	1.3760×10^{-4}	β_h	+tve	1.0000			
τ_c	-tve	0.3703						
τ_h	-tve	7.6831×10^{-6}						
θ_c	+tve	1.0000						
θ_h	+tve	9.9708×10^{-5}						
β_c	+tve	0.9999						
β_h	+tve	9.9708×10^{-5}						

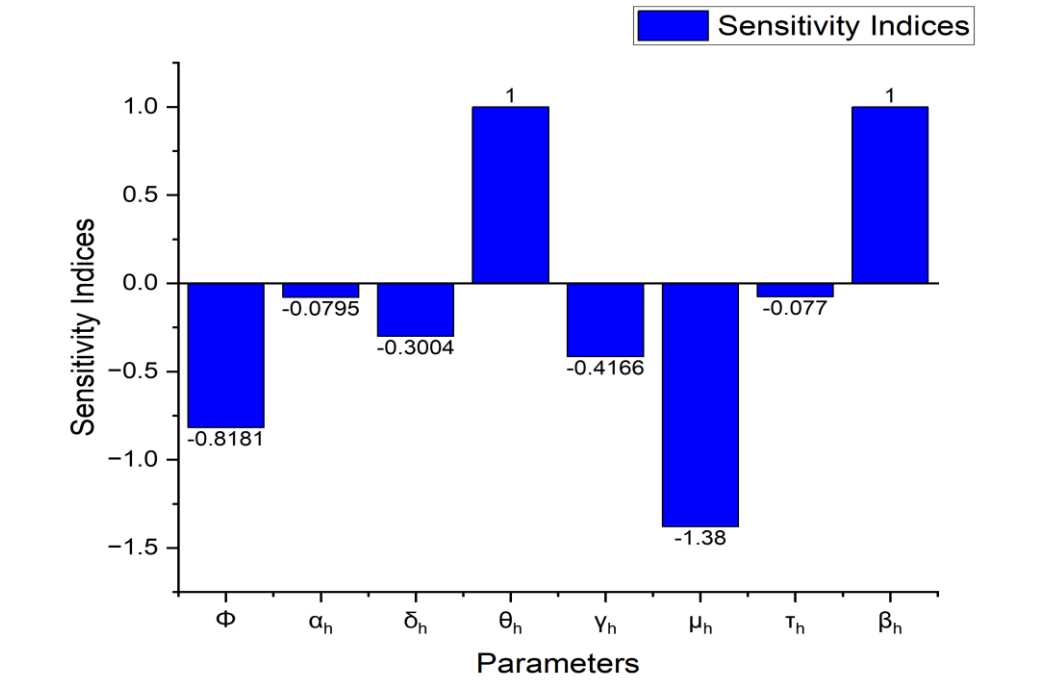


Figure 2: Sensitivity Indices chart for Human

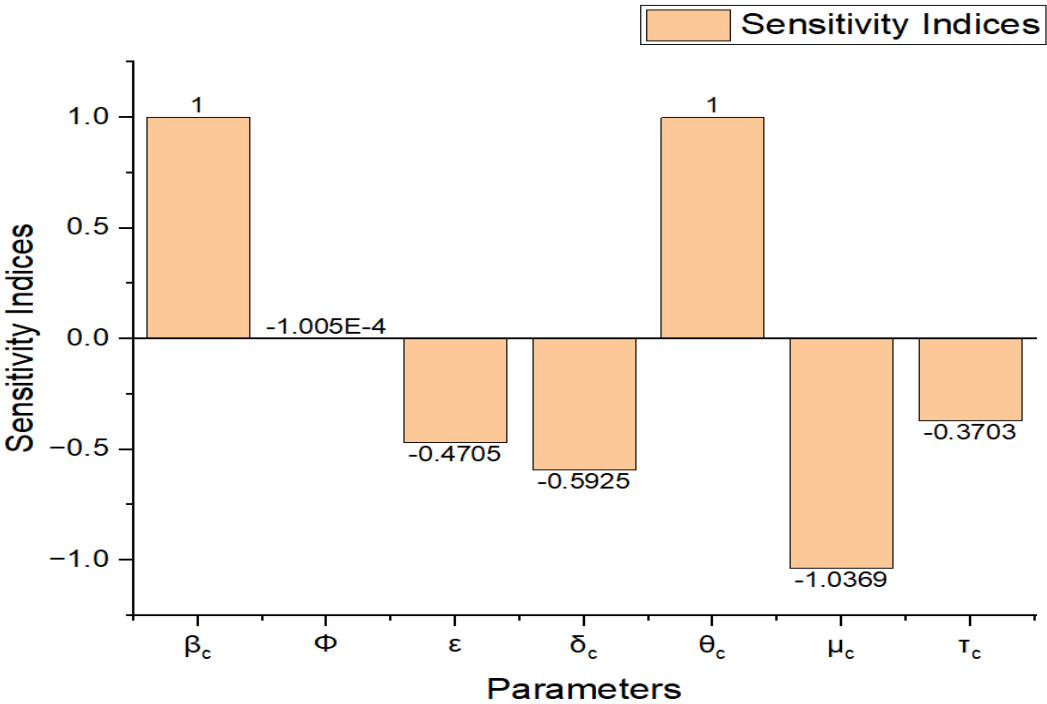


Figure 3: Sensitivity Indices chart for Cattle

The sensitivity analysis of the model parameters was carried out using OriginPro 2024 software, which was also employed to generate the corresponding sensitivity charts.

The detailed sensitivity analysis of R_{0H} , and R_{0c} showed that increasing (decreasing) β_h , θ_h and β_c , θ_c would increase (decreasing) R_{0H} , and R_{0c} , respectively. And increasing ϕ , α_h , δ_h , γ_h , μ_h , τ_h and ε , δ_c , μ_c , τ_c would decrease R_{0H} , and R_{0c} , respectively and vice versa. Therefore, to mitigate the spread of the Anthrax, it is recommended to cut the cattle infection rate and keep the human infection probability low.

Optimal Control of the Anthrax Model

The essence of optimal control in disease modelling is to determine the best control strategy in fighting the spread of the infection and the costs associated with this strategy. The analysis is carried out to establish the impact of various intervention schemes. Optimal control problem is established by introducing controls into the anthrax model (3) and the formation of an objective functional which seeks to minimize (u_1, u_2, u_3, u_4) .

In this framework, $u_1(t)$ represents human preventive measures (education, PPE, use, avoidance of infected animal or products). $u_2(t)$ Corresponds to early treatment of detected human cases (antibiotics for I_{ED}). $u_3(t)$ is the intensive treatment for late human cases (hospitalization of I_{LD}). Lastly, $u_4(t)$ involves the vaccination efficacy of susceptible cattle. we define the objective function as

$$J = \min_{(u_1, u_2, u_3, u_4)} \int_{t_0}^{t_1} \left[A_1 I_{ED} + A_2 I_{LD} + A_3 I_c + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2) \right] dt \quad (32)$$

Subject to the state equations above, with appropriate state initial conditions. t_0 is the initial time and t_1 is the terminal time. The constant terms $A_1, A_2, A_3, B_1, B_2, B_3, B_4$ represent the weight constants. The terms

$B_1 u_1^2, B_2 u_2^2, B_3 u_3^2, B_4 u_4^2$ the costs associated with human preventive measures (education, PPE, use, avoidance of infected animal or products), early treatment of detected human cases (antibiotics for I_{ED}), intensive treatment for late human cases (hospitalization of I_{LD}), and vaccination efficacy of susceptible cattle. The control set is defined as:

$$U = \{(u_1, u_2, u_3, u_4) | 0 \leq u_1 \leq u_{1\max}, 0 \leq u_2 \leq u_{2\max}, 0 \leq u_3 \leq u_{3\max}, 0 \leq u_4 \leq u_{4\max}\}$$

Pontryagin's Maximum Principle

The principle gives the necessary conditions that an optimal must be satisfied . It changes the system in (3) and (32) into minimisation problem pointwise Hamiltonian,(H) with respect to (u_1, u_2, u_3, u_4) .

Theorem 2.1. There exist optimal controls $(u_1^*, u_2^*, u_3^*, u_4^*)$ and corresponding solutions, $(S_h, I_{ed}, I_{ld}, R_h, V_c, S_c, I_c, R_c)$ that minimize $J(u_1, u_2, u_3, u_4)$ over U.

Proof. The integrand of the objective functional J given by (32) is a convex function of (u_1, u_2, u_3, u_4) and the state system (3) satisfies the Lipschitz property to the state variables since state solutions are bounded. To find an optimal solution, we need to find the Lagrangian and Hamiltonian for the problem (3). The Lagrangian of the control problem is given by

$$L = A_1 I_{ED} + A_2 I_{LD} + A_3 I_c + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2)$$

Introducing the co-state $\lambda_1, \dots, \lambda_8$ associated with $(S_h, I_{ed}, I_{ld}, R_h, V_c, S_c, I_c, R_c)$ respectively, is given by

$$\begin{aligned} H = & A_1 I_{ED} + A_2 I_{LD} + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + B_4 u_4^2) + \lambda_1 (\theta_h - (1-u_1) \beta_h \Gamma S_h - \mu_h S_h) \\ & + \lambda_2 ((1-u_1) \beta_h \Gamma S_h - (\alpha_h + \gamma_h + u_2 + \mu_h) I_{ED}) + \lambda_3 (\alpha_h I_{ED} - (\mu_h + \delta_h + \tau_h + u_3) I_{LD}) \\ & + \lambda_4 (\tau_h I_{LD} + \gamma_h I_{ED} + u_3 I_{LD} - \mu_h R_h) + \lambda_5 (u_4 \theta_c \varepsilon - (\omega + \mu_h) V_c) + \lambda_8 (\tau_c I_c - \mu_c R_c) \\ & + \lambda_6 (u_4 \theta_c (1-\varepsilon) + \omega V_c + (1-u_1) \beta_c \Gamma S_c - \mu_c S_c) + \lambda_7 ((1-u_1) \beta_c \Gamma S_c + (\tau_c + \delta_c + \mu_c) I_c) \end{aligned} \quad (33)$$

where $\lambda_i, i = 1, 2, 3, \dots, 8$ are the adjoint variables.

The solutions of adjoint are system;

$$\begin{aligned} \frac{d\lambda_1}{dt} &= \lambda_1 ((1-u_1) \beta_h (1-\phi) (I_c + I_{ED} + I_{ED}) + \mu_h) - \lambda_2 (1-u_1) \beta_h (1-\phi) (I_c + I_{ED} + I_{ED}), \\ \frac{d\lambda_2}{dt} &= -A_1 + \lambda_1 (1-u_1) \beta_h (1-\phi) S_h - \lambda_2 ((1-u_1) \beta_h (1-\phi) S_h - \mu_h - \alpha_h - \gamma_h - u_2) - \lambda_3 \alpha_h \\ &\quad - \lambda_4 \gamma_h + \lambda_6 (1-u_1) \beta_c (1-\phi) S_c - \lambda_7 (1-u_1) \beta_c (1-\phi) S_c, \end{aligned}$$

$$\begin{aligned} \frac{d\lambda_3}{dt} = & -A_1 + \lambda_1(1-u_1)\beta_h(1-\phi)S_h - \lambda_2(1-u_1)\beta_h(1-\phi)S_h + \lambda_3(\mu_h + \alpha_h + \gamma_h + u_2) \\ & - \lambda_4(\tau_h + u_3) + \lambda_6(1-u_1)\beta_c(1-\phi)S_c - \lambda_7(1-u_1)\beta_c(1-\phi)S_c, \end{aligned}$$

$$\frac{d\lambda_4}{dt} = \lambda_4\mu_h, \quad (34)$$

$$\frac{d\lambda_5}{dt} = \lambda_4(\mu_c + \omega) - \lambda_6\omega,$$

$$\frac{d\lambda_6}{dt} = \lambda_6((1-u_1)\beta_c(1-\phi)(I_C + I_{ED} + I_{ED}) + \mu_c) - \lambda_7(1-u_1)\beta_c(1-\phi)(I_C + I_{ED} + I_{ED}),$$

$$\begin{aligned} \frac{d\lambda_7}{dt} = & \lambda_1(1-u_1)\beta_h(1-\phi)S_h - \lambda_2(1-u_1)\beta_h(1-\phi)S_h + \lambda_6(1-u_1)\beta_c(1-\phi)S_c \\ & - \lambda_7((1-u_1)\beta_c(1-\phi)S_c - \mu_c - \delta_c - \tau_c) - \lambda_8\tau_c, \end{aligned}$$

$$\frac{d\lambda_8}{dt} = \lambda_8\mu_c.$$

which satisfies the transversality condition;

$$\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 = \lambda_6 = \lambda_7 = \lambda_8 = 0 \quad (35)$$

The optimal control vector $(u_1^*, u_2^*, u_3^*, u_4^*)$ that maximizes the objective function J over U , is given by;

$$\begin{aligned} u_1^* &= \max \left\{ 0, \min \left(\frac{((\lambda_7 - \lambda_6)\beta_c S_c + (\lambda_2 - \lambda_1)\beta_h S_h)\Gamma}{B_1}, 1 \right) \right\} \\ u_2^* &= \max \left\{ 0, \min \left(\frac{\lambda_2 I_{ED}}{B_2}, 1 \right) \right\} \\ u_3^* &= \max \left\{ 0, \min \left(\frac{(\lambda_3 - \lambda_4)I_{LD}}{B_3}, 1 \right) \right\} \\ u_4^* &= \max \left\{ 0, \min \left(\frac{((\lambda_6 - \lambda_5)\varepsilon - \lambda_6)\theta_c}{B_4}, 1 \right) \right\} \end{aligned} \quad (36)$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8$ are the solutions of equations (34) and (35).

Numerical Results and Discussions

In this section, we'll explore some numerical examples of model (1) to see how different control strategies both individually and combined impact the outcomes. We'll start with initial values like $S_h(0) = 800$, $I_{cd}(0) = 20$, $I_d(0) = 10$, $R_h(0) = 50$, $V_c(0) = 100$, $S_c(0) = 600$, $I_c(0) = 50$, and $R_c(0) = 30$. The implemented strategies are outlined, and graphical illustrations of the model are provided using MATLAB 2023 in the figures below to facilitate clearer visualization of the results.

Strategy A: Implementation of no control

$$u_1 = 0, u_2 = 0, u_3 = 0, u_4 = 0$$

Strategy B: Implementation of single control

$$u_1 \neq 0, u_2 = 0, u_3 = 0, u_4 = 0$$

$$u_1 = 0, u_2 \neq 0, u_3 = 0, u_4 = 0$$

$$u_1 = 0, u_2 = 0, u_3 \neq 0, u_4 = 0$$

$$u_1 = 0, u_2 = 0, u_3 = 0, u_4 \neq 0$$

Strategy C: Implementation of double controls

$$u_1 \neq 0, u_2 \neq 0, u_3 = 0, u_4 = 0$$

$$u_1 \neq 0, u_2 = 0, u_3 \neq 0, u_4 = 0$$

$$u_1 \neq 0, u_2 = 0, u_3 = 0, u_4 \neq 0$$

$$u_1 = 0, u_2 \neq 0, u_3 \neq 0, u_4 = 0$$

$$u_1 = 0, u_2 \neq 0, u_3 = 0, u_4 \neq 0$$

$$u_1 = 0, u_2 = 0, u_3 \neq 0, u_4 \neq 0$$

Strategy D: Implementation of three controls

$$u_1 \neq 0, u_2 \neq 0, u_3 \neq 0, u_4 = 0$$

$$u_1 \neq 0, u_2 \neq 0, u_3 = 0, u_4 \neq 0$$

$$u_1 \neq 0, u_2 = 0, u_3 \neq 0, u_4 \neq 0$$

$$u_1 = 0, u_2 \neq 0, u_3 \neq 0, u_4 \neq 0$$

Strategy E: Implementation four controls

$$u_1 \neq 0, u_2 \neq 0, u_3 \neq 0, u_4 \neq 0$$

Strategy B: For one control only

The three figures demonstrate that implementing a 90% effective control measure targeting human and cattle ($u_1 = 0.9$) effectively eliminates disease transmission across all populations, whereas the uncontrolled scenario results in severe epidemic outcomes. In Figure 1a, (I_c), the control strategy prevents the characteristic sharp outbreak peak of approximately 162 infected cattle observed without intervention, instead driving immediate suppression and near-elimination within 5–7 days. This reservoir control cascades to human populations: Figure 1b, (I_{cd}) shows that without control, early-detected human infections follow a concerning biphasic pattern with an initial massive surge (approximately 430 cases), a secondary wave (approximately 115 cases), and eventual endemic persistence (approximately 100 cases), whereas with control, infections remain negligible and rapidly

disappear. Similarly, Figure 1c, (I_{ld}) reveals that late-detected human infections exhibit parallel dynamics peaking at approximately 110 cases with subsequent reintroduction and endemic stabilization (approximately 35 cases) without control, but remaining minimal and quickly eliminated when the cattle reservoir is managed. Collectively, these results indicate that targeting the zoonotic source alone is sufficient to prevent both the initial epidemic explosion and secondary waves in humans, achieving disease elimination without requiring direct human-targeted interventions.

Strategy C: For any two controls:

The six dual-control scenarios reveal that early antibiotic treatment (u_2) is the critical non-negotiable component for effective epidemic management, while the combination of human and cattle prevention without frontline treatment can be counterproductive. In figure 2(a): human prevention plus early treatment) achieved near-elimination of both early and late infections by rapidly clearing cases before progression, whereas figure 2(b) failed due to delayed resurgence from lack of early intervention, and figure 2(c) paradoxically produced worse outcomes than the uncontrolled baseline by shifting the epidemic into a larger secondary wave. Scenarios incorporating u_2 with either cattle vaccination (u_4) or hospitalization (u_3) significantly reduced peaks (figure 2(e)) but could not fully prevent resurgence without the synergistic protection of human preventive measures (u_1). Conversely, strategies lacking early treatment particularly figure 2(f) resulted in the highest initial peaks and persistent endemic states, demonstrating that for Anthrax diseases with human transmission, clinical management at the early detection stage must accompany vaccination programs, as treating the human reservoir is as essential as controlling the animal source.

Strategy D: For the three controls; human preventive measure, early and late treatment of detected human:

The three figures demonstrate that a comprehensive human-centric intervention strategy combining 90% preventive measures (u_1), 100% early antibiotic treatment (u_2), and intensive hospitalization (u_3) achieves complete anthrax elimination across both species without any cattle vaccination. In Figure 3(a), the strategy indirectly suppresses the cattle reservoir from an initial 50 cases to elimination within days, despite no veterinary intervention, by breaking the anthrax transmission chains. Figure 3(b) shows total eradication of the characteristic biphasic human epidemic preventing both the massive initial surge (approximately 430 cases) and secondary wave observed in the uncontrolled baseline, with

infections decaying from approx.15 to zero by day 5. Similarly, Figure 3(b) eliminates late detected infections, with the initial approximation of 10 cases rapidly cleared through the synergistic action of early treatment preventing progression and hospitalization managing any advanced cases. This demonstrates that maximum-intensity human intervention alone can achieve cross-species elimination, rendering direct cattle vaccination unnecessary when prevention and frontline clinical management operate at optimal efficacy.

Strategy E: For all the four controls; human preventive measure, early treatment of detected human, late treatment of detected human and Vaccination:

The three figures demonstrate that implementing all four control measures simultaneously 90% human preventive measures (u_1), 100% early antibiotic treatment (u_2), intensive hospitalization (u_3), and cattle vaccination (u_4) achieve immediate disease elimination across cattle and human populations, though with notable redundancy compared to human-only strategies. Figure 4(a) shows rapid suppression of cattle infections from approx. 50 to zero within days, while Figures 4(b) and 4(c) reveal complete eradication of both early and late human infections, with minimal initial cases decaying to zero by day 5 and no secondary waves emerging. Critically, comparison with the previous three-control scenario (without cattle vaccination) reveals identical elimination trajectories, indicating that when human interventions operate at maximum efficacy, direct veterinary intervention becomes superfluous the animal reservoir is controlled indirectly through broken interspecies transmission chains. This suggests that resource allocation should prioritize comprehensive human control, reserving cattle vaccination for scenarios with limited human intervention capacity or when animal health protection is an independent objective beyond epidemic suppression.

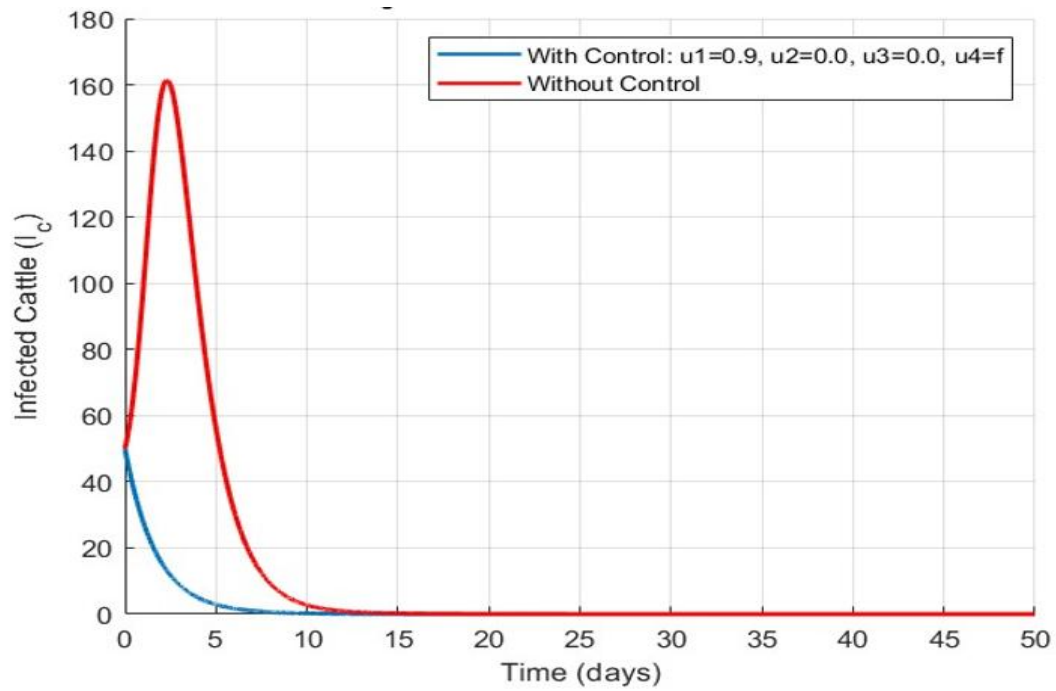


Figure 1a: Graph showing the effect of human preventive measures on Infected Cattle

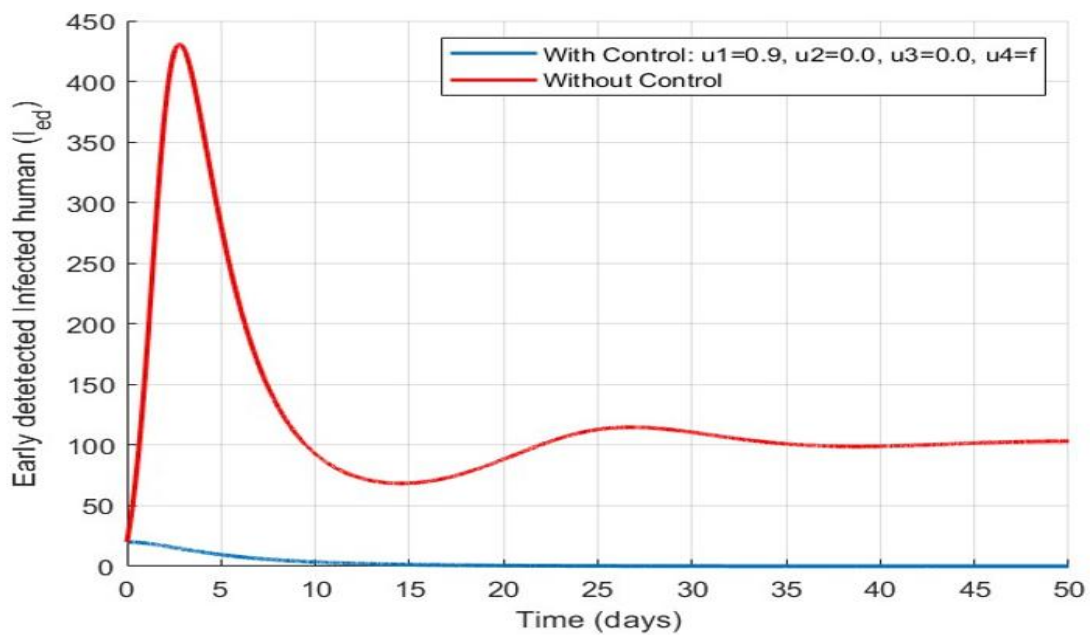


Figure 1b: Dynamics of human preventive measures on early detected infected human

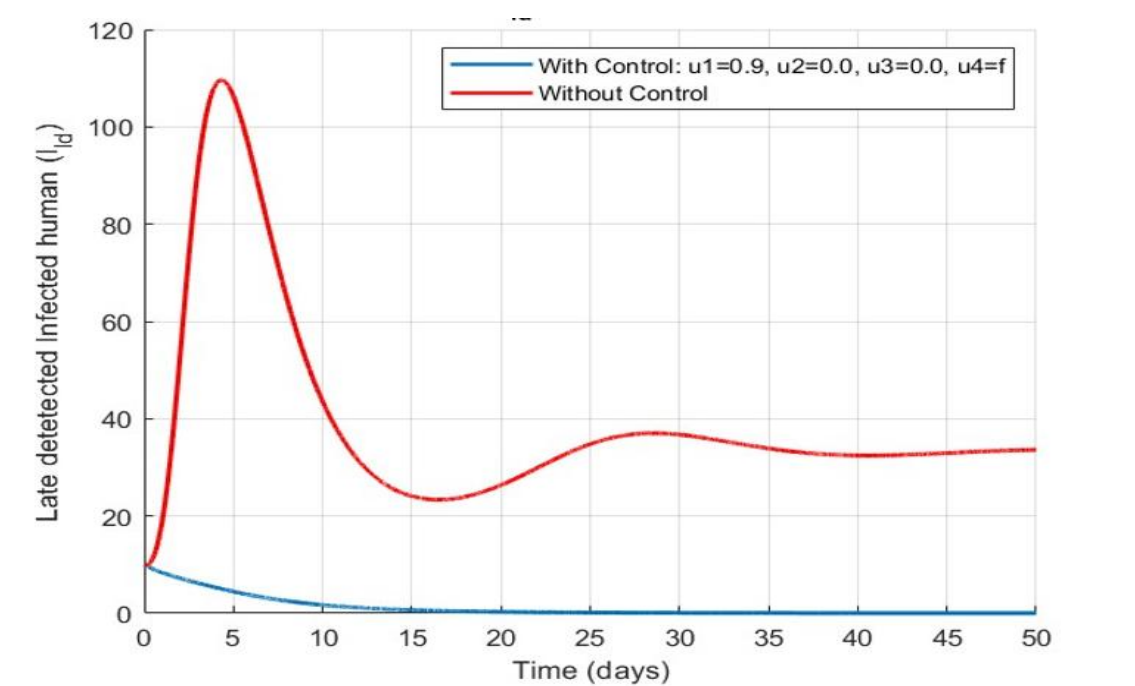


Figure 1b: Dynamics of human preventive measures on late detected infected human

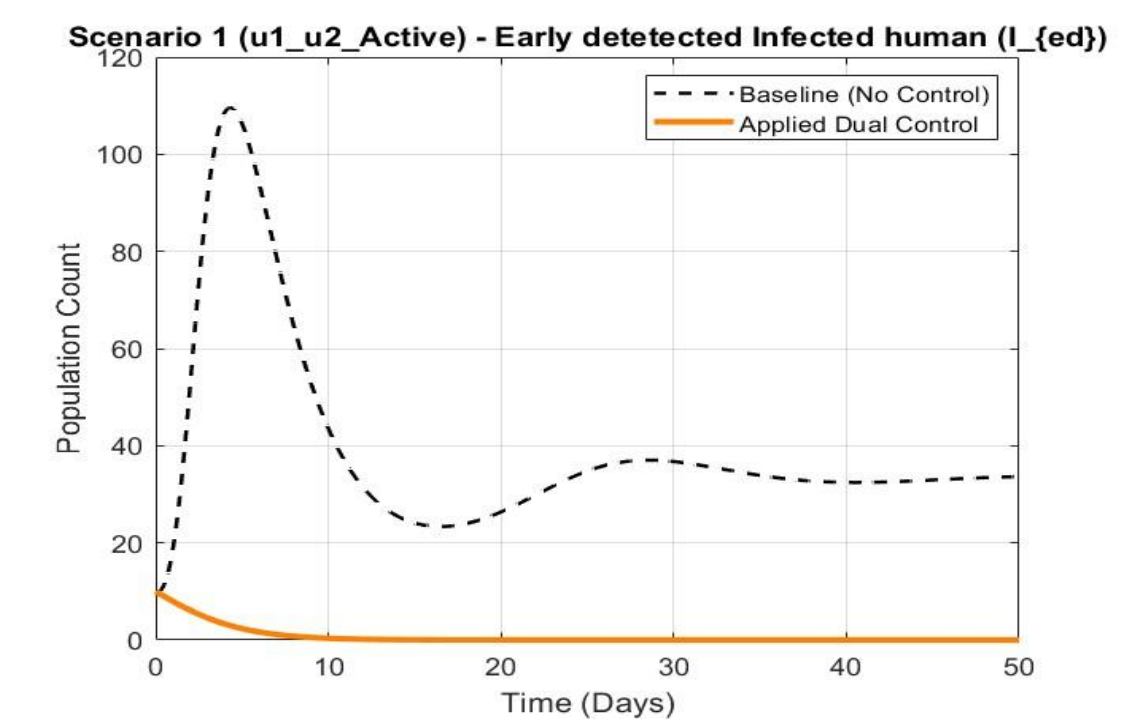


Figure 2a: Dynamics of dual Control Combination (u_1 and u_2) on early detected infected human

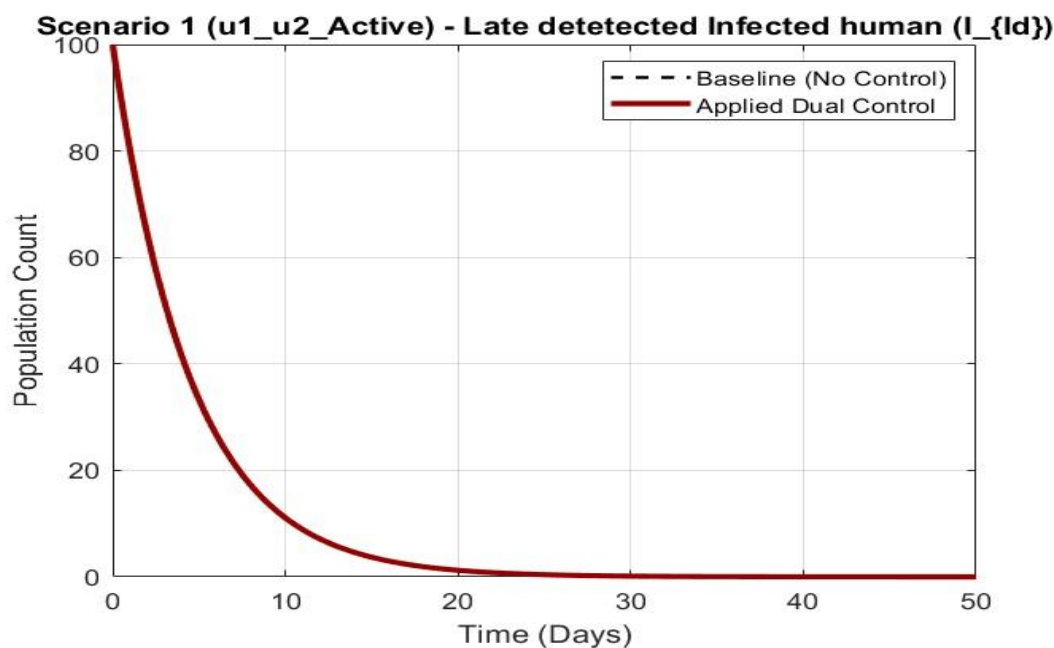


Figure 2b: Dynamics of dual Control Combination (u_1 and u_2) on late detected infected human

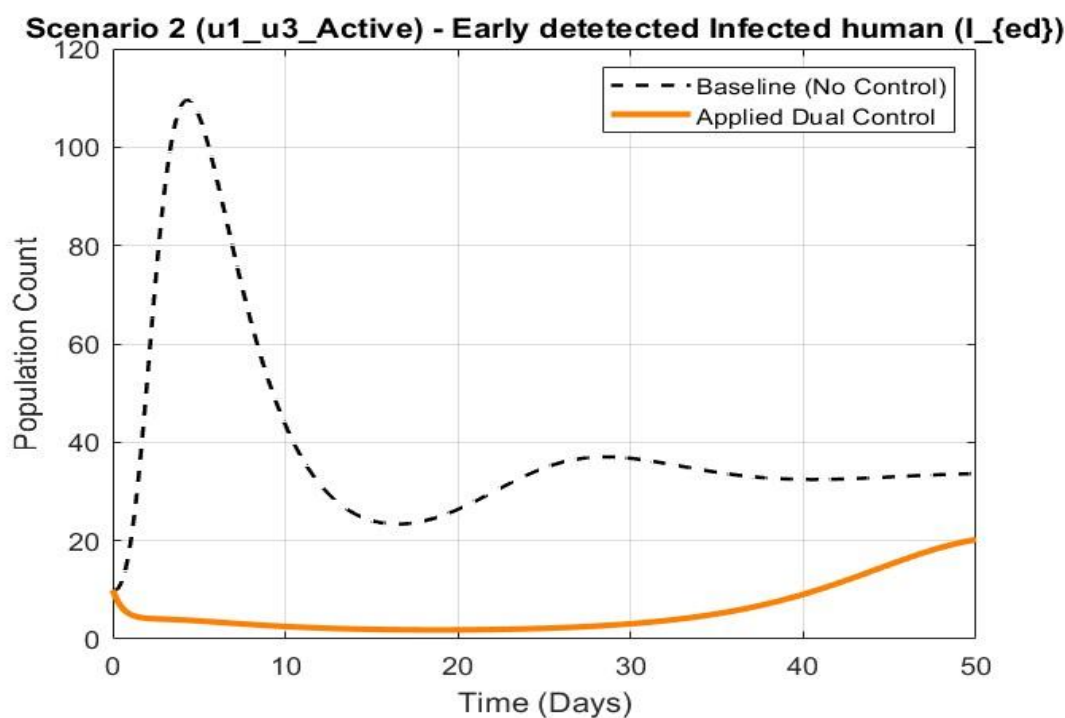


Figure 2c: Dynamics of dual Control Combination (u_1 and u_3) on early detected infected human

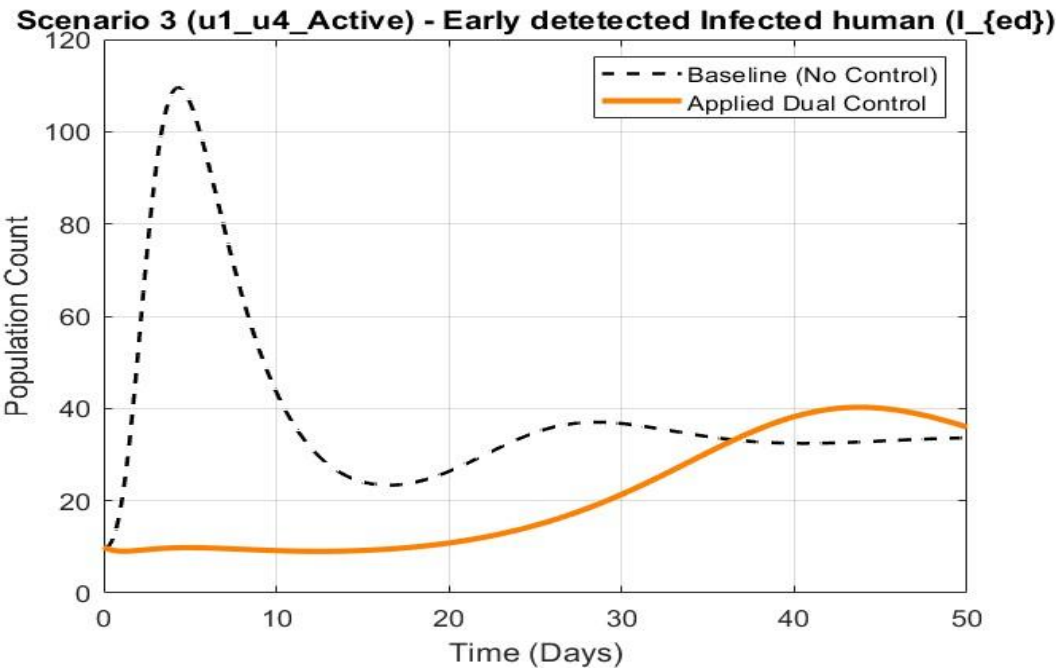


Figure 2d: Dynamics of dual Control Combination (u_1 and u_4) on early detected infected human

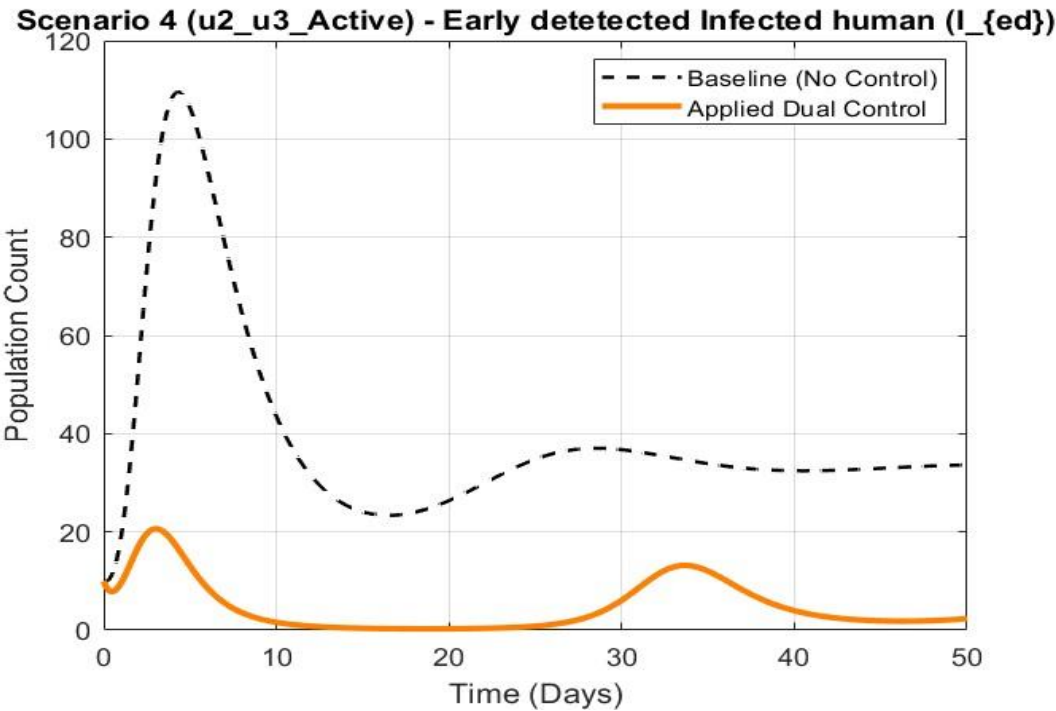


Figure 2e: Dynamics of dual Control Combination (u_2 and u_3) on early detected infected human

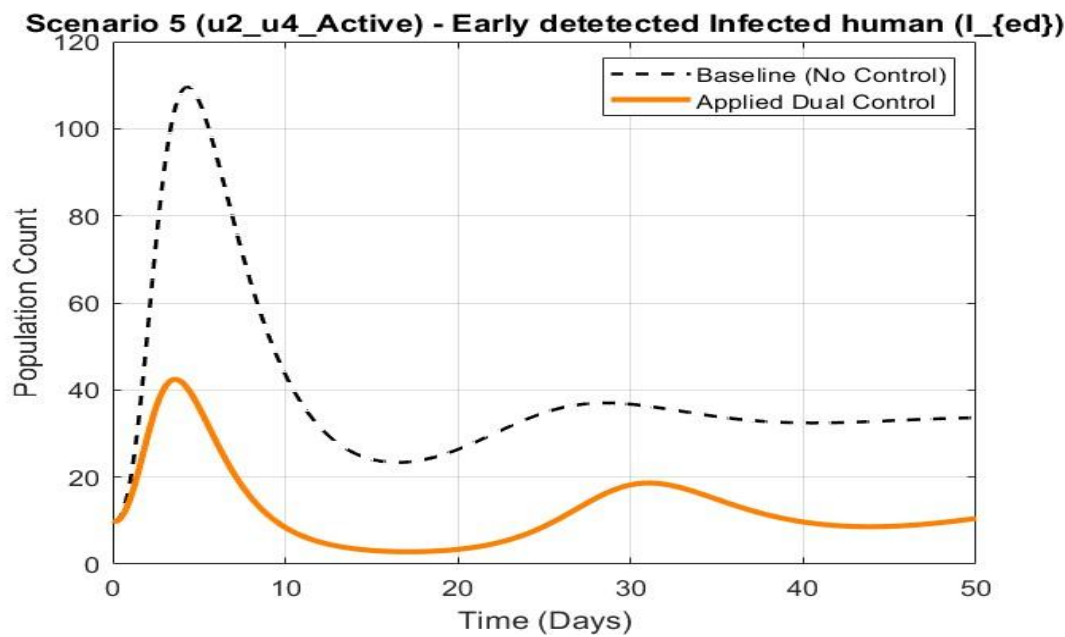


Figure 2f: Dynamics of dual Control Combination (u_2 and u_4) on early detected infected human

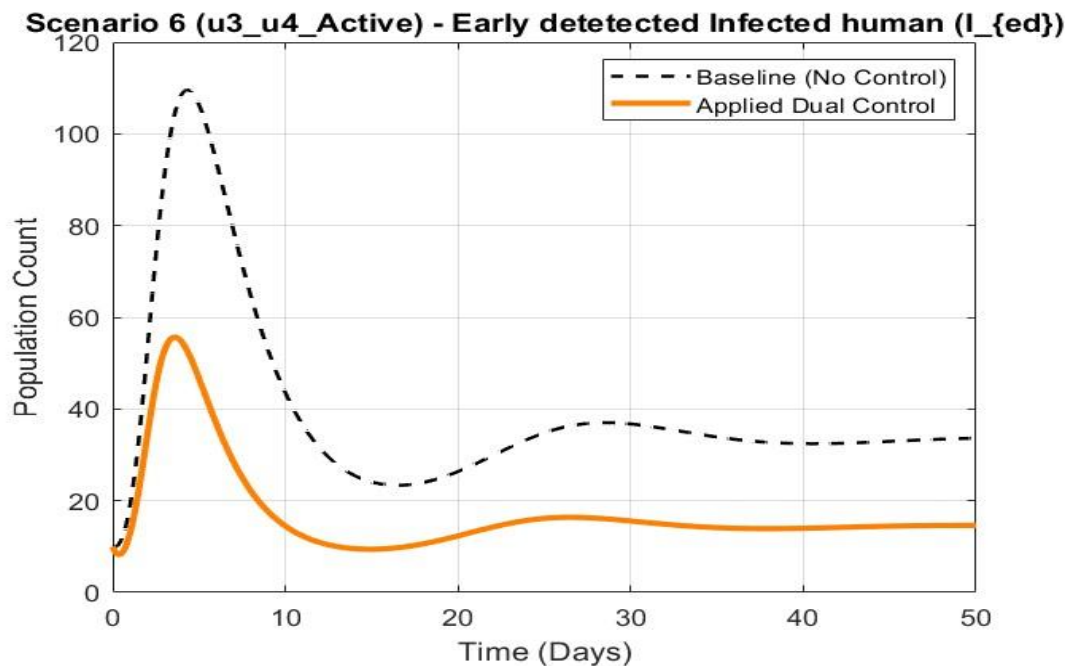


Figure 2g: Dynamics of dual Control Combination (u_3 and u_4) on early detected infected human

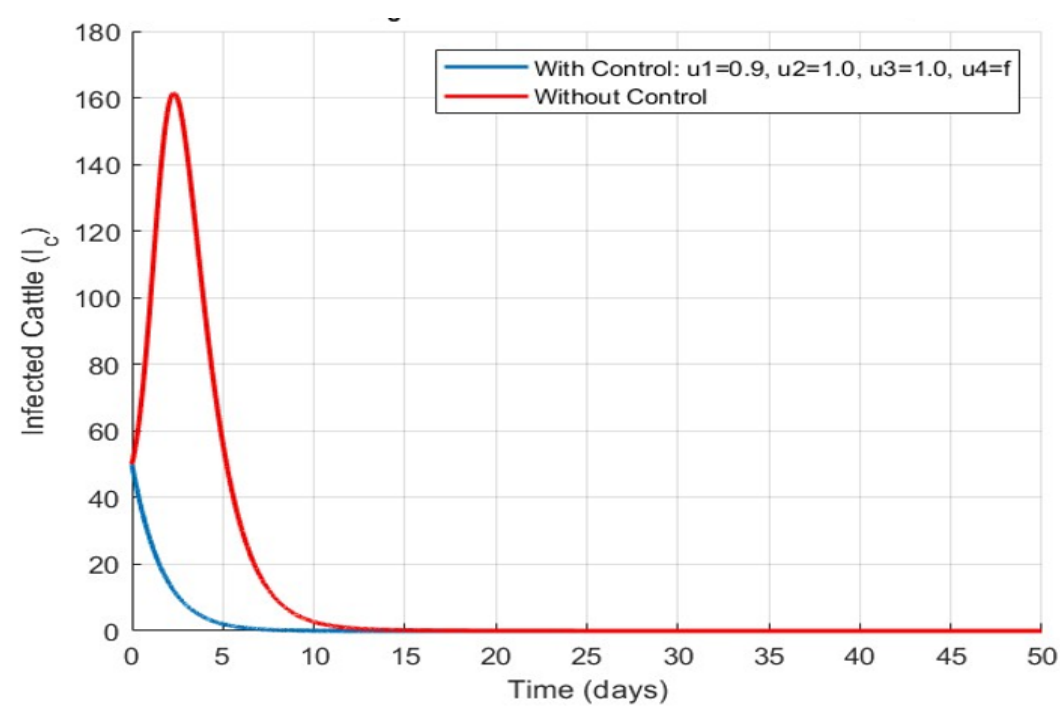


Figure 3a: Dynamics of three Control Combination (u_1, u_2 and u_3) on infected cattle

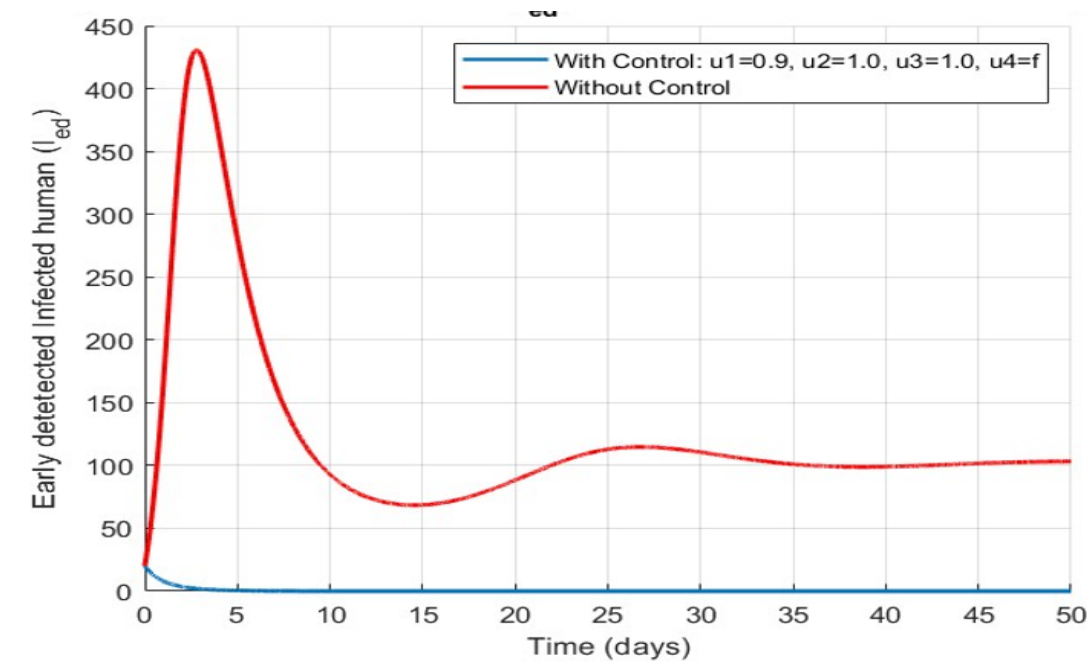


Figure 3b: Dynamics of three Control Combination (u_1, u_2 and u_3) on early detected infected human

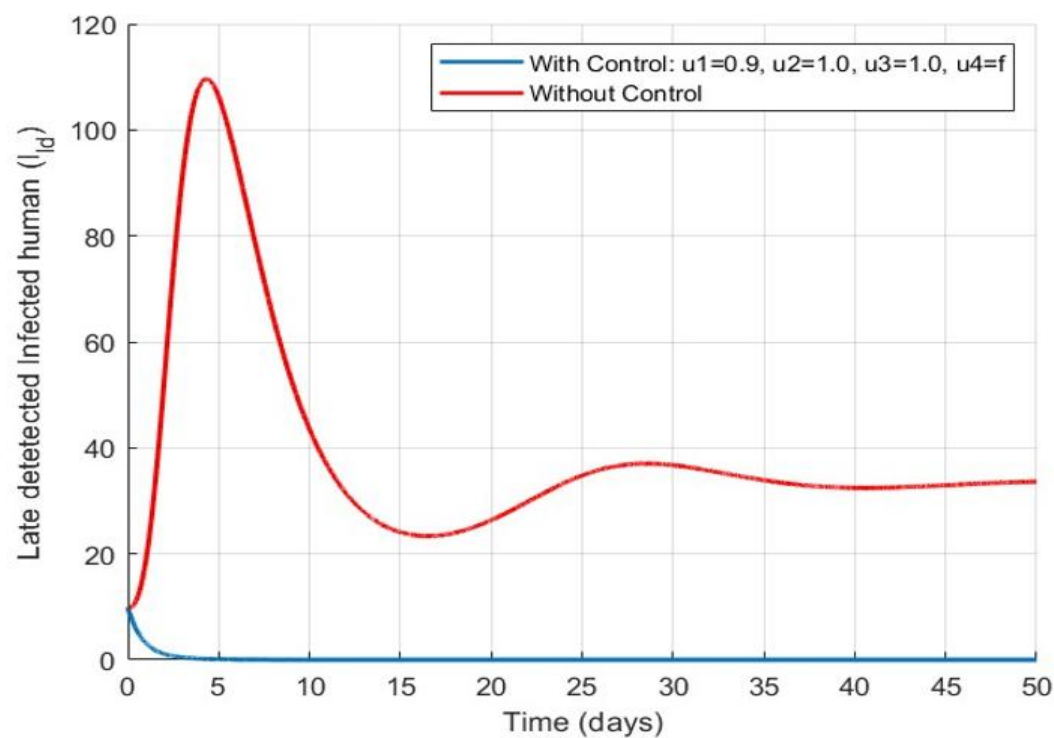


Figure 3c: Dynamics of three Control Combination (u_1 , u_2 and u_3) on late detected infected human

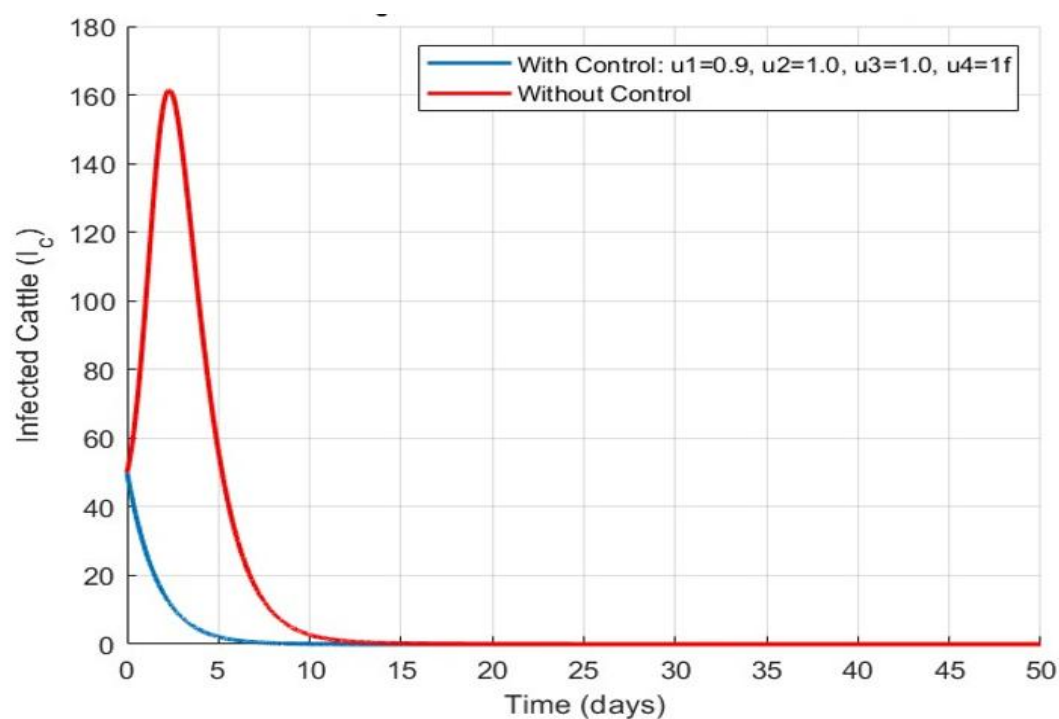


Figure 4a: Dynamics of four Control Combinations (u_1 , u_2 , u_3 and u_4) on infected cattle

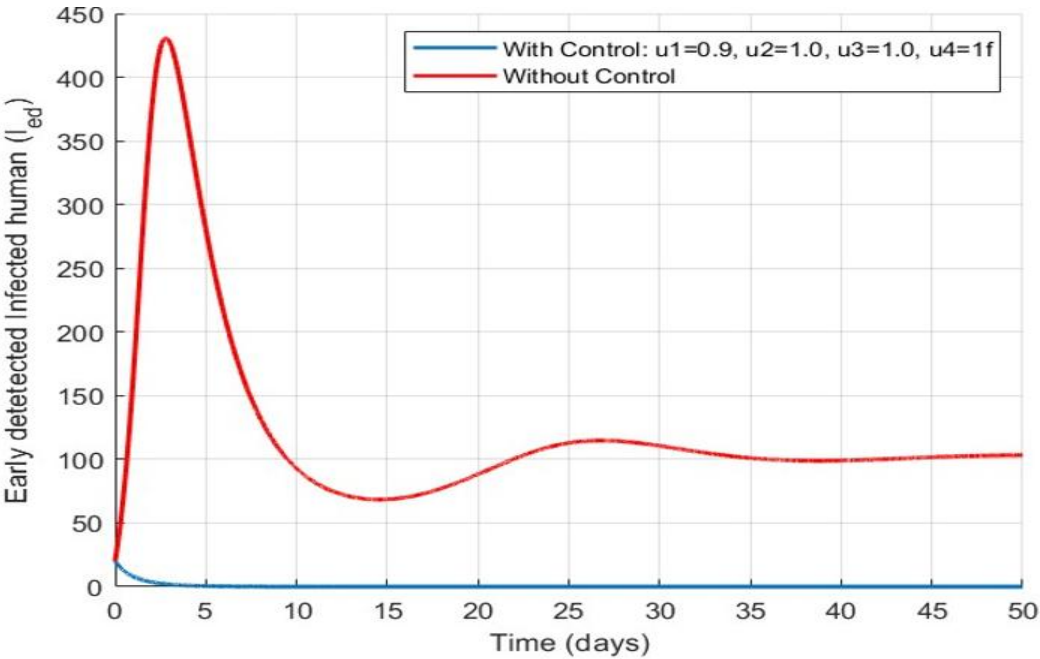


Figure 4b: Dynamics of four Control Combinations (u_1 , u_2 , u_3 and u_4) on early detected infected human

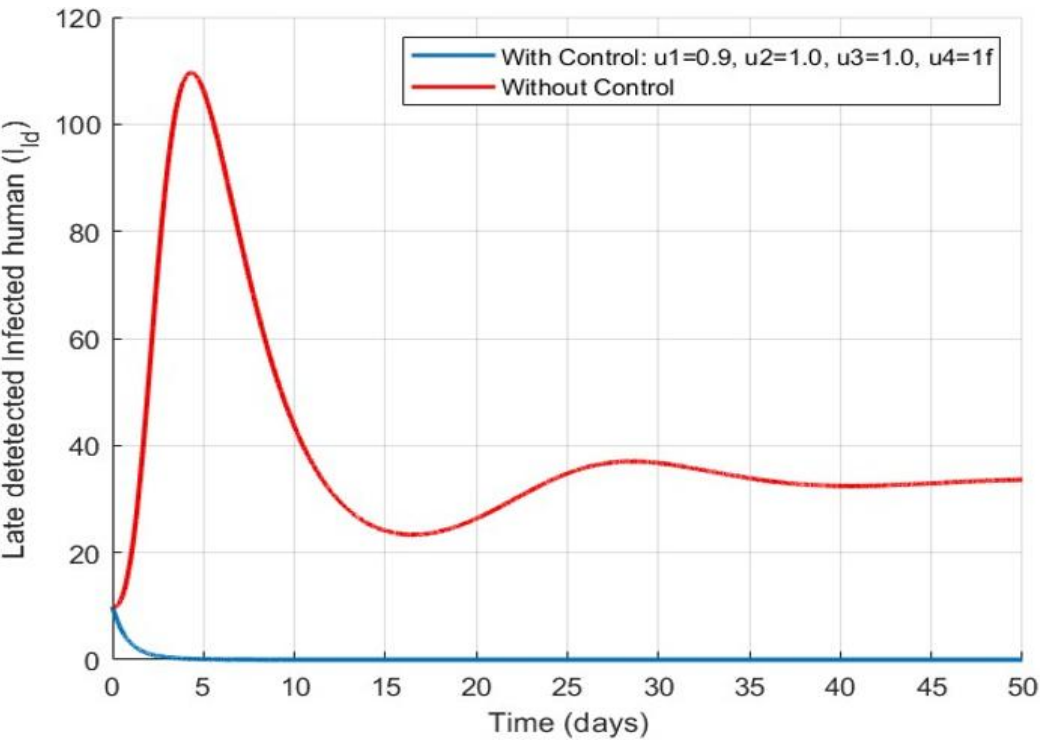


Figure 4c: Dynamics of four Control Combinations (u_1 , u_2 , u_3 and u_4) on late detected infected human

CONCLUSION

This study demonstrates that effective control of anthrax transmission requires a combination of animal vaccination and early human intervention strategies. The analysis shows that reducing infection rates in cattle and minimizing human exposure are critical for limiting disease spread.

Optimal control results further reveal that prompt antibiotic treatment in humans plays a vital role in eliminating the disease, and when combined with preventive measures, provides the most cost-effective strategy. Although multiple control measures can achieve eradication, their added benefits diminish over time, making the integrated approach of prevention and early treatment the most efficient and practical solution for managing anthrax outbreaks.

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