Journal of Science Research and Reviews
PRINT ISSN: 1595-9074
E-ISSN: 1595-8329
DOI: https://doi.org/10.70882/josrar.2025.v2i3.94
Homepage: https://josrar.esrgngr.org
Original Research Article

Mathematical Model for the Dynamics of Anthrax in Human and Animal Populations Incorporating Control Measures

[1]Abubakar, Abdulrahman, [1]Bala, Maigari J., [1]Umar, Ahmed G., *[2]Abdullahi, Musa and [3]Gidado, Umar

[1]Department of Mathematics Education, Adamawa State Polytechnic, Yola, Nigeria
[2]Department of Mathematics, Modibbo Adama University, Yola, Nigeria
[3]Government Day Senior Secondary School Hammawa Toungo, Yola, Nigeria
*Corresponding Author's email: mabdallah@mau.edu.ng

KEYWORDS
Anthrax,
Reproduction Number,
Vaccination,
Stability,
Simulations.

CITATION
Abubakar, A., Bala, M. J., Umar, A. G., Abdullahi, M., & Gidado, U. (2025). Mathematical Model for the Dynamics of Anthrax in Human and Animal Populations Incorporating Control Measures. *Journal of Science Research and Reviews*, *2*(3), 166-177. https://doi.org/10.70882/josrar.2025.v2i3.94

ABSTRACT
Anthrax is a zoonotic infectious disease caused by the bacterium *Bacillus anthracis*. In this study, we develop and analyze a deterministic compartmental model, formulated using ordinary differential equations, to explore the transmission dynamics of anthrax between humans and animals. Fundamental properties of the model, such as positivity, boundedness, and the existence of equilibrium points are established, confirming that the model is mathematically and biologically well-posed. The basic reproduction number, R_0, is derived using the next-generation matrix approach. Stability analysis shows that the disease-free equilibrium is both locally and globally asymptotically stable when $R_0 < 1$. Additionally, the model possesses a unique endemic equilibrium when $R_0 > 1$, which is also globally stable when $R_0 < 1$. Numerical simulations are conducted to validate and illustrate the theoretical results.

INTRODUCTION

Bacterium *Bacillus anthracis* is the cause of the infectious disease called anthrax. It usually affects both domestic and wild animals and is found naturally in soil (Samad, 2013). The sole obligatory aerobic pathogen in the genus Bacillus is the gram-positive, rod-shaped, non-motile bacterium known as *Bacillus anthracis*. The bacterium can exist as a long-lasting, extremely resistant spore form or as a vegetative form. In the host's low-oxygen environment, the bacteria only survive in the vegetative state (Ahmed, *et al.,* 2010). In difficult conditions, the anthrax Bacillus develops spores to protect itself. The thick outer layer of spores protects the bacteria from harsh environments (Decker, 2003).

Although anthrax seldom affects birds, it can infect cats and dogs who eat meat contaminated with the disease; these animals frequently recover from anthrax without medical intervention (Decker, 2003; Stoltenow, 2021). It is important to remember that not every animal with anthrax dies. An animal's innate immunity and the amount of infections it has been exposed to are likely to determine whether it survives or not (Decker, 2003).

Once inside its host, an anthrax spore germinates, becomes a vegetative cell, and multiplies quickly. Following their entry into the bloodstream, the reproduced vegetative cells cause septicemia, which ultimately results in the host's abrupt death (Dragon *et al.,* 1995). The extended half-life of anthrax spores allows them to survive for decades in the environment before they find a new host. Spores are typically seen in areas where corpses infected with anthrax have been decomposing for a long time. It has been documented that spores can re-infect animals more than 70 years after disturbed burial sites containing anthrax-infected animals (Awoonor-Williams, 2016).

Humans can contract anthrax from contaminated animal products or from diseased animals themselves. The way

anthrax enters the body determines the kind of disease that a person gets. Anthrax usually enters the body by the gastrointestinal tract, lungs, or skin. If left untreated, anthrax of any kind has the potential to gradually spread throughout the body and result in death (Nigeria Center for Disease Control NCDC, 2023). Numerous factors, such as the source of infection, affect the likelihood that humans may get anthrax. The source of anthrax infection includes cutaneous, gastrointestinal and inhalation anthrax (Fasanella *et al.,* 2014).

Scientists and epidemiologists have found that using mathematical models to investigate the transmission and management of infectious diseases is a valuable resource (Hethcote, 2000). This research seeks to use mathematical models to study the trans mission dynamics of anthrax in animal and human population by modifying the model due to Baloba and Seidu (2022) by incorporate vaccination and exposure as compartments in the animal population and also educated, un educated and individuals under treatment as compartments in the human population.

Mathematical modeling has played a crucial role in improving the understanding and control of infectious diseases like anthrax. These analytical tools have been valuable in forecasting disease trends and guiding healthcare professionals in developing effective management strategies. A wide range of mathematical models has been formulated and examined to explore the transmission dynamics of anthrax in human and animal populations (Suma, *et al.,* 2018; Yedata *et al.,* 2020; Elijah *et al.,* 2020). These studies have highlighted key factors influencing the spread of the disease and have proposed various control strategies.

Mushayabasa (2015) added a set time delay and environmental decontamination to Hahn and Furniss's (1983) model on the eradication of anthrax illness. Using the reproduction number, they investigated how well environmental decontamination eradicated the anthrax pathogen. The concept of Mushayabasa (2015) was expanded by Sinkie and Murthy (2016), who postulated that certain infectious animals may exhibit clinical symptoms of the illness and be treated to recover. Their research demonstrated that when the rate of cure is accelerated through therapy, the pathogen level dropped and the number of vulnerable animals rose.

Shaibu, Oluwole and David (2018) developed a mathematical model for the disease's patterns of transmission. The mathematical model was used to formulate ordinary differential equations. They carried out the model's quantitative and qualitative study to explain the anthrax disease's patterns of transmission. They examined and ascertained the steady state solutions for the model. The anthrax model's disease-free equilibrium 7 was examined for locally asymptotic stability and the corresponding epidemic basic reproduction number. When the basic reproductive number is smaller than unity, the model's disease-free equilibrium has been demonstrated to be locally asymptotically stable. Additionally, population lowers the environmental pathogen level. the broader public's shift in behavior. The model looks at anthrax in both human and animal populations, and the findings indicated that an increase in the basic reproduction number would result from a decrease in the animal recovery rate. Furthermore, it would raise the fundamental reproduction number by raising the pace of human recruitment. Furthermore, a decline in the rates of human recruitment, animal recruitment, livestock transmission, and human transmission would result in a reduction of the basic reproduction number. There would be an increase in the basic reproduction number if the rates of human recruitment, animal recruitment, livestock transmission, and human transmission all increased.

Efraim *et al.,* (2018) proposed a deterministic model for anthrax transmission dynamics in humans and animals, analyzing and determining which parameters drive the disease transmission dynamics. The basic reproduction number was calculated, and a sensitivity index for each parameter in the basic reproduction number was determined. The findings indicate that animal recruitment and infection rates are more vulnerable to disease transmission. Anthrax infection grows when more animals are recruited, and reduces as animals die naturally. Their findings are supported by numerical simulations employing the Runge-Kutta method, which suggest that animals influence the dynamics of anthrax. To remove the disease, the study offers control techniques such as animal immunization, fumigation, and carcass decomposition.

Osman *et al.*, (2018) developed a mathematical model of anthrax in both human and animal populations, building on Friedman and Yakubu's (2013) model. The model was expanded to optimal control, taking into account preventive strategies for susceptible humans, animal vaccination, and treatment for infected humans and animals. Both qualitative and quantitative analyses were explored. The findings indicated that animal immunization and human prevention are the most effective strategies for pre venting anthrax epidemics.

Motivated by aforementioned studies, this work aims to modify the model presented in Baloba and Seidu (2022) vaccine for susceptible animals, public health campaign (education) and treatment for susceptible humans as control measures in the face of exposure to the bacteria for both humans and animals. The structure of this paper is as follows: Section 2 presents the model formulation, including the underlying assumptions, the flow diagram, model equations, and their basic properties. Section 3 provides the analytical results, covering the equilibrium points, the basic reproduction number, R_0, and the

stability analysis of the equilibria. In Section 4, we outline the parameter values used to compute the basic reproduction number, and display related numerical simulations. Finally, Section 5 discusses the findings and concludes the study.

MATERIALS AND METHODS

Model formulation

Two populations coexisting in the same environment make up the modified model: human and animal. At any time t, The total human population denoted by $N_H(t)$ is split into six subpopulations of susceptible educated, $S_e(t)$, susceptible uneducated $S_u(t)$, exposed humans $E_h(t)$, infected humans $I_h(t)$), treated humans, $T_h(t)$ and recovered humans $R_h(t)$, so that:

$$N_h(t) = S_e(t) + S_u(t) + E_h(t) + I_h(t) + T_h(t) + R_h(t) \quad (1)$$

At each given time t, the animal population denoted by $N_a(t)$ is divided into five sub-populations that are susceptible animals $S_a(t)$, exposed animals $E_a(t)$, infected animals $I_a(t)$, recovered animals $R_a(t)$, and vaccinated animals $V_a(t)$. Hence, the total number of animal population is given by

$$N_a(t) = S_a(t) + E_a(t) + I_a(t) + R_a(t) + V_a(t) \quad (2)$$

The susceptible educated human population $S_e(t)$ is increase by public health enlightenment of humans at a rateθ_h, and the population of humans who recovered from anthrax after losing infection acquired immunity at a rate π_h . This population is de creased by natural death μ_h, and force of infection at a rate $(1-\tau)\lambda_h$ where τ measure the efficacy of the public health education and $\lambda_h = \frac{\beta_h(P+I_a)}{1+\kappa_h I_h}$, where β_h is the human transmission rate, such that

$$\frac{dS_e}{dt} = \theta_h S_u + \pi_h R_h - (\mu_h + (1-\tau)\lambda_h)S_e \quad (3)$$

Also recruitment into susceptible uneducated human population $S_u(t)$ is by birth at a rate Λ_h and the population is decreased by natural deaths, public health enlightenment of human, and force of infection at a rate μ_h, θ_h and λ_h, respectively. so that

$$\frac{dS_u}{dt} = \Lambda_h - (\lambda_h + \mu_h + \theta_h)S_u \quad (4)$$

The exposed human population $E_h(t)$ is increased by the force of infection from both educated and un-educated class at a rate $(1-\tau)\lambda_h S_e$ and $\lambda_h S_u$ respectively, it is decreased by μ_h and ε_h where μ_h is rate of natural death and ε_h is the rate at which the latent human progresses to the infected class. Such that

$$\frac{dE_h}{dt} = (1-\tau)\lambda_h S_e + \lambda_h S_u - (\mu_h + \varepsilon_h)E_h \quad (5)$$

The infected humans population $I_h(t)$ is increased by the rate at which the latent human progresses to the infected class at a rate ε_h, and decreased by natural death, rate at which infected humans move to treatment class and death due to anthrax disease at a rate μ_h, ϕ_h and C_h respectively. so that

$$\frac{dI_h}{dt} = \varepsilon_h E_h - (\mu_h + \phi_h + C_h)I_h \quad (6)$$

The infected humans under treatment population $T_h(t)$ is increased by treatment at a rate ϕ_h, and decreased by natural death a rate μ_h and the rate at which humans recover from anthrax at the rate of δ_h respectively. so that

$$\frac{dT_h}{dt} = \phi_h I_h - (\mu_h + \delta_h)T_h \quad (7)$$

The Recovered humans population $R_h(t)$ is increased by recovery rate due to treatment at a rate δ_h. The class is decreased by natural death rate and the rate at which recovered humans from anthrax revert to susceptible educated human at a rate μ_h and π_h and πh respectively. so that

$$\frac{dR_h}{dt} = \delta_h T_h - (\mu_h + \pi_h)R_h \quad (8)$$

The compartment P is increased by infected animals shed anthrax pathogens into the environment at a rate ξ_a and reduced due to environmental hygiene at rate p and by natural phenomenon at rate η respectively. So that

$$\frac{dP}{dt} = \xi_a I_a - (p+\eta)P \quad (9)$$

Similarly, susceptible animal population $S_a(t)$ is increased by animal annual birth rate Λ_a, waning of vaccination acquired immunity in animals at a rate α_a and the population of animal that recovered from anthrax after losing infection acquired immunity at a rate π_a, the population is decreased by administering vaccination in animals, natural death, and force of infection at a rate $\gamma\gamma_a, \mu_a$ and $\lambda_a = \frac{\beta_a P}{1+\kappa_a I_a}$ respectively. so that

$$\frac{dS_a}{dt} = \Lambda_a + \pi_a R_a + \alpha_a V_a - (\lambda_a + \mu_a)S_a - \gamma_a S_a \quad (10)$$

The exposed animal population $E_a(t)$ is increased by the force of infection at a rate $\lambda_a S_a$, and is decreased by natural death and the rate at which the exposed animal joint the infected class at a rate μ_a and ε_a respectively. so that

$$\frac{dE_a}{dt} = \lambda_a S_a - (\mu_a + \varepsilon_a)E_a \quad (11)$$

The infected animal population $I_a(t)$ is increased by the progression of exposed animal to infected class at the rate ε_a, and is decreased by natural death, the progression of infected animal to the recovered class and the death due to anthrax disease at the rate μ_a, δ_a and C_a respectively. so that

$$\frac{dI_a}{dt} = \varepsilon_a E_a - (\mu_a + C_a + \delta_a)I_a \quad (12)$$

The Recovered animals population $R_a(t)$ is increased by recovery rate due to treatment at a rate δ_a. The class is further decreased by natural death and rate at which recovered animal from anthrax revert to susceptible animals at the rate μ_a and π_a respectively. so that

$$\frac{dR_a}{dt} = \delta_a I_a - (\mu_a + \pi_a)R_a \quad (13)$$

The vaccinated animals population $V_a(t)$ is increased by vaccine administered to susceptible animals at a rate γ_a, it is decreased by natural death and waning of vaccination in animals at a rate μ_a and α_a respectively. so that

$$\frac{dV_a}{dt} = \gamma_a S_a - (\alpha_a + \mu_a)V_a \quad (14)$$

The above model was formulated based on the following assumptions:

1. All newly born individuals are recruited into an un educated susceptible class.
2. Recovered individuals are allowed to move into educated susceptible class.
3. Newly born and other susceptible animals are vaccinated.
4. There is an incubation period for both animals and humans after infection.
5. There is failure in vaccinations in animal population so that vaccinated populations revert to susceptible.
6. The parameter $\tau \in [0,1]$ is considered to quantify the success of the public health campaign (education) in reducing the population's anthrax infection. If $\tau = 0$, the public health campaign (education) has no effect on the behavior of susceptible individuals. However, if $\tau = 1$, the public health campaign (education) is 100% effective in improving the behavior of susceptible individuals towards taking protective measures against anthrax disease.

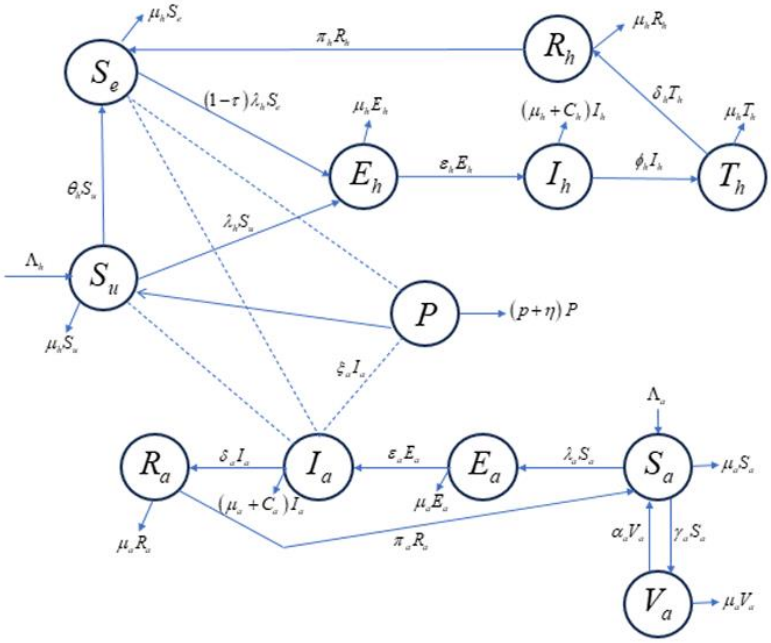

Figure 1: Schematic Diagram of the Model

Below is the system of equations defining the dynamics of anthrax

$$\left.\begin{aligned}
\frac{dS_e}{dt} &= \theta_h S_u + \pi_h R_h - (\mu_H + (1-\tau)\lambda_h) S_e \\
\frac{dS_u}{dt} &= \Lambda_h - (\lambda_h + \mu_H + \theta_h) S_u \\
\frac{dE_h}{dt} &= (1-\tau)\lambda_h S_e + \lambda_h S_u - (\mu_h + \varepsilon_h) E_h \\
\frac{dI_h}{dt} &= \varepsilon_h E_h - (\mu_H + \phi_h + C_h) I_h \\
\frac{dT_h}{dt} &= \phi_h I_h - (\mu_h + \delta_h) T_h \\
\frac{dR_h}{dt} &= \delta_h T_h - (\mu_h + \pi_h) R_h \\
\frac{dP}{dt} &= \xi_a I_a - (p+\eta) P \\
\frac{dS_a}{dt} &= \Lambda_a + \pi_a R_a + \alpha_a V_a - (\lambda_a + \mu_a) S_a - \gamma_a S_a \\
\frac{dE_a}{dt} &= \lambda_a S_a - (\mu_a + \varepsilon_a) E_a \\
\frac{dI_a}{dt} &= \varepsilon_a E_a - (\mu_a + C_a + \delta_a) I_a \\
\frac{dR_a}{dt} &= \delta_a I_a - (\mu_a + \pi_a) R_a \\
\frac{dV_a}{dt} &= \gamma_a S_a - (\alpha_a + \mu_a) V_a
\end{aligned}\right\} \quad (15)$$

With the force of infection being $\lambda_h = \frac{\beta_h (P + I_a)}{1 + \kappa_h I_h}$ and $\lambda_a = \frac{\beta_a P}{1 + \kappa_a I_a}$ respectively.

Table 1: State variables of the model

State Variable	Description
S_e	Susceptible educated human population at time t
S_u	Susceptible un-educated human population at time t
E_h	Exposed human population at time t
I_h	Infected human population at time t
T_h	Treated human population at time t
R_h	Recovered human population at time t
P	Environmental reservoir contaminated with anthrax pathogens at time t
S_a	Susceptible animal population at time t
E_a	Exposed animal population at time t
I_a	Infected animal population at time t
R_a	Recovered human population at time t
V_a	Vaccinated human population at time t

Table 2: Parameter description of the model

Parameter	Description
Λ_h/Λ_a	Human recruitment rate/inflow rate of animals
μ_h, μ_a	Natural death rate of humans/animals
β_h, β_a	Transmission rate in humans/animals
λ_h/λ_a	Force of infection in humans/animals
C_h/C_a	Disease induced death rate in humans/animals
τ	Public health education efficacy
θ_h	Rate at which uneducated susceptibles receive public health education
ε_h	Rate of progression from exposed to infected humans
ε_a	Rate of progression from exposed to infected animals
α_a	Rate of waning of vaccine in animals
ξ_a	Pathogen shedding rate
η	Rate of environmental pathogens' natural degradation
κ_a	The population saturation effect of animals
κ_h	Rate of behavioural change
p	Rate of decay due to environmental hygiene
γ_a	Rate at which animals are vaccinated
ϕ_h	Treatment rate of humans
δ_a	Recovery rate of infected animals
δ_h	Human recovery rate after treatment
π_a	Rate at which animals that have recovered return to susceptibles
π_h	Rate at which recovered humans revert to susceptibles

Basic properties of the model

In this section, both the qualitative and quantitative outcomes of the model is presented. We proved that the model solution exists and is unique, as well as that it is positive and bounded. Since the model system (1) monitors human and dog populations, all its associated parameters are non-negative. Further, the following non-negativity result holds:

Theorem 1

The variables of the model system (1) are non-negative for all time $t > 0$. In other words, the solution of the model system (1) with positive initial data will remain positive for all time $t > 0$.

Proof: Let

$t_1 = sup\{t > 0: S_e > 0, S_u > 0, E_h \geq 0, I_h \geq 0, T_h \geq 0, R_h \geq 0, P \geq 0, S_a > 0, E_a \geq 0, I_a \geq 0, R_a \geq 0, V_a \geq 0\}$.

Thus, $t_1 > 0$. It follows from the first equation of model system (2.1) that:

$\frac{dS_e}{dt} = \theta_h S_u + \pi_h R_h - (\mu_h + (1-\tau)\lambda_h)S_e \geq -(\mu_h + (1-\tau)\lambda_h)S_e$,

So that

$$\frac{dS_e}{dt} + (\mu_h + (1-\tau)\lambda_h)S_e \geq 0 \quad (16)$$

Multiplying the inequality (2) by the integrating factor, $\rho(t) = exp\left[\mu_h t + \int_0^t (1-\tau)\lambda_h(u)du\right]$ gives;

$$\rho(t)\left[\frac{dS_e}{dt} + (\mu_h + (1-\tau)\lambda_h)S_e\right] = \frac{d(S\rho)}{dt} \geq 0 \quad (17)$$

from (17) it follows that $S_e(t) \geq 0$ for all $t \geq 0$. Using the positivity of $S_e(t)$, it can, similarly, be shown that the remaining state variables of the model (1) are non-negative

(for all non-negative initial conditions) for $t \geq 0$. Consequently, all the solutions of the model (1), with non-negative initial conditions, remain non-negative for all time $t \geq 0$.
We claim the following result.

Theorem 2
Let $(S_e, S_u, E_h, I_h, T_h, R_h, P, S_a, V_a, E_a, I_a, R_a)$ be the solution of the model system (1) with non-negative initial conditions. The closed set

$$\Omega = \left\{X = \mathbb{R}_+^{12} \middle| N_h \leq \frac{\Lambda_h}{\mu_h}, N_a \leq \frac{\Lambda_a}{\mu_a}, P \leq \frac{\xi_a \Lambda_a}{\mu_a(p+\eta)}\right\} \quad (18)$$

is bounded and positively invariant and attracting with respect to the model (1) where
$\mathbb{R}_+^{12} = [S_u, S_e, E_h, I_h, T_h, R_h, P, S_a, V_a, E_a, I_a, R_a]$
Proof: From the Equations of the total populations $N_h(t), N_a(t)$ and P we have the following:

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$$
$$\frac{dN_a}{dt} \leq \Lambda_a - \mu_a N_a \quad (19)$$

We construct the proof by applying the theorem on differential inequality to equation (19) gives

$$N_h \leq \frac{\Lambda_h}{\mu_h}[1 - e^{-\mu_h t}] + N_h(0)e^{-\mu_h t} \quad (20)$$

and

$$N_a \leq \frac{\Lambda_a}{\mu_a}[1 - e^{-\mu_a t}] + N_a(0)e^{-\mu_a t} \quad (21)$$

Thus, the size of the human population $N_h \to \frac{\Lambda_h}{\mu_h}$ as $t \to \infty$, and the size of animal population $N_a \to \frac{\Lambda_a}{\mu_a}$ as $t \to \infty$. Hence, all solutions of model system (15) are contained in the region Ω. Thus, Ω is bounded.

Model Analysis

Existence and stability of anthrax-free equilibrium

The anthrax-free equilibrium point is a steady-state solution in which no disease spreads throughout the population. The disease-free equilibrium point of the system is achieved when all variables and parameters linked to anthrax infection are zero. Thus, $E_h = 0, I_h = 0, R_h = 0, P = 0, E_a = 0, R_a = 0, \text{and } I_a = 0.$
Therefore, the anthrax-free equilibrium point denoted by E_0 is given as:

$$\Gamma_0 = [S_{e0}, S_{u0}, 0,0,0,0, S_{a0}, 0,0,0, V_{a0}] \quad (22)$$

where:

$$S_{e0} = \frac{\Lambda_h \theta_h}{\mu_h(\mu_h+\theta_h)}, S_{u0} = \frac{\Lambda_h}{\mu_h+\theta_h},$$
$$S_{a0} = \frac{\Lambda_a(\alpha_a+\mu_a)}{\mu_a(\alpha_a+\mu_a+\gamma_a)}, V_{a0} = \frac{\Lambda_a \gamma_a}{\mu_a(\alpha_a+\mu_a+\gamma_a)}$$

Using the next-generation matrix method, the disease-free equilibrium of model (15) given in (22) is locally asymptotically stable if the spectral radius of matrix FV^{-1} is less than one. Let $X = (E_h, I_h, T_h, P, E_a, I_a)$ which can be written in the form of $\frac{dX}{dt} = F_i(x) - V_i(x)$, where

$$F_i = \begin{bmatrix} (1-\tau)\frac{\beta_h(P+I_a)}{1+\kappa_h I_h} S_e + \frac{\beta_h(P+I_a)}{1+\kappa_h I_h} S_u \\ 0 \\ 0 \\ 0 \\ \frac{\beta_a P}{1+\kappa_a I_a} \\ 0 \end{bmatrix}, \quad \text{and}$$

$$V_i = \begin{bmatrix} (\mu_h + \varepsilon_h)E_h \\ -\varepsilon_h E_h + (\mu_h + \phi_h + C_h)I_h \\ -\phi_h I_h + (\mu_h + \delta_h)T_h \\ -\xi_a I_a + (p+\eta)P \\ (\mu_a + \varepsilon_a)E_a \\ -\varepsilon_a E_a + (\mu_a + C_a + \delta_a)I_a \end{bmatrix}$$

we define the reproduction number of the model as:

$$R_0 = \rho(FV^{-1}) = \frac{\beta_h \varepsilon_h \Lambda_h(\mu_h + \theta_h(1-\tau))}{\mu_h(\mu_h+\theta_h)(\mu_h+\varepsilon_h)(C_h+\mu_h+\phi_h)} \quad (23)$$

The basic reproduction number, denoted by R_0, for model (15) represents the average number of new anthrax infections (in either humans or animals) generated by a single infectious animal introduced into a population where both humans and animals are entirely susceptible. We claim the following result.

Theorem 3

The disease-free equilibrium (DFE) of model (15) is locally asymptotically stable whenever $R_0 < 1$, indicating that anthrax cannot invade the population. Conversely, the DFE is unstable if $R_0 > 1$, meaning the infection can spread in the population.

Epidemiological Implication of Theorem 3

Theorem 3 implies that if the basic reproduction number $R_0 < 1$, the introduction of a small number of infected animals into a well-mixed human-animal population will not result in a significant anthrax outbreak, provided the initial number of infected humans or animals remains low. This suggests that under such conditions, the disease will eventually die out without causing widespread transmission.

Global stability of anthrax-free equilibrium point

We investigate the global stability of Anthrax free equilibrium for the model equation (15) using the conditions of Castillo-Chavez and Song (2004). First, equation (15) must be written in the form

$$\frac{dY}{dt} = F(Y, Z)$$
$$\frac{dZ}{dt} = G(Y, Z); G(Y, Z) = 0 \quad (24)$$

where $Y = (S_e, S_u, R_h, S_a, V_a, R_a)$ which denotes the number of uninfected compartments with components $Y \in \mathbb{R}^6$ and $Z = (E_h, I_h, T_h, P, E_a, I_a)$ denotes the number of infected compartments with components $Z \in \mathbb{R}^6$ with E_0 given in (22). The two conditions to be met for global asymptotic stability are:

1. $\frac{dY}{dt} = F(Y, 0), E_0$ is globally asymptotically stable (GAS).

2. $\hat{G}(Y,Z) = AZ - G(Y,Z) \geq 0$ for $(X,Z) \in \Omega$

$A = D_j G(Y,0)$ is an M-Matrix (the off diagonal elements of A are all non-negative) and Ω is a feasible region.

Condition 1

From model equations (1), we have

$$F(Y,Z) = \begin{bmatrix} \theta_h S_u + \pi_h R_h - \left(\mu_h + (1-\tau)\frac{\beta_h(P+I_a)}{1+\kappa_h I_h}\right) S_e \\ \Lambda_h - \left(\frac{\beta_h(P+I_a)}{1+\kappa_h I_h} + \mu_h + \theta_h\right) S_u \\ \delta_h T_h - (\mu_h + \pi_h) R_h \\ \Lambda_a + \pi_a R_a + \alpha_a V_a - \left(\frac{\beta_a P}{1+\kappa_a I_a} + \mu_a + \gamma_a\right) S_a \\ \gamma_a S_a - (\alpha_a + \mu_a) V_a \\ \delta_a I_a - (\mu_a + \pi_a) R_a \end{bmatrix} \quad (25)$$

Evaluating (25) at E_0, we have

$$F(Y,0) = \begin{bmatrix} \theta_h S_u^0 - \mu_h S_e^0 \\ \Lambda_h - (\mu_h + \theta_h) S_u^0 \\ 0 \\ \Lambda_a + \alpha_a V_a^0 - (\mu_a + \gamma_a) S_a^0 \\ \gamma_a S_a^0 - (\alpha_a + \mu_a) V_a^0 \\ 0 \end{bmatrix} \quad (26)$$

Therefore E_0 is globally asymptotically stable.

Condition 2

$\hat{G}(Y,Z) = AZ - G(Y,Z), G(Y,Z) \geq 0$ for $(Y,Z) \in \Omega$, now

$$G(Y,Z) = \begin{bmatrix} (1-\tau)\frac{\beta_h(P+I_a)}{1+\kappa_h I_h} S_e + \frac{\beta_h(P+I_a)}{1+\kappa_h I_h} S_u - (\mu_h + \varepsilon_h) E_h \\ \varepsilon_h E_h - (\mu_h + \phi_h + C_h) I_h \\ \phi_h I_h - (\mu_h + \delta_h) T_h \\ \xi_a I_a - (p+\eta) P \\ \frac{\beta_a P}{1+\kappa_a I_a} S_a - (\mu_a + \varepsilon_a) E_a \\ \varepsilon_a E_a - (\mu_a + C_a + \delta_a) I_a \end{bmatrix} \quad (27)$$

and $A = D_j(Y,0)$ is the Jacobian of $G(Y,Z)$ with respect to Z, such that

$$A = \begin{bmatrix} -K_1 & y_1 & 0 & -y_1 & 0 & y_2 \\ \varepsilon_h & -K_2 & 0 & 0 & 0 & 0 \\ 0 & \phi_h & -K_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & -K_4 & 0 & \xi_a \\ 0 & 0 & 0 & \frac{\beta_a S_a}{1+\kappa_a I_a} & -K_5 & \frac{\beta_a P S_a \kappa_a}{(1+\kappa_a I_a)^2} \\ 0 & 0 & 0 & 0 & \varepsilon_a & -K_6 \end{bmatrix} \quad (28)$$

where:

$$K_1 = \mu_h + \varepsilon_h, K_2 = \mu_h + \phi_h + C_h, K_3 = \mu_h + \delta_h, K_4 = p + \eta, K_5 = \mu_a + \varepsilon_a, K_6 = \mu_a + \delta_a + C_a$$

$$y_1 = \frac{(1-\tau)\beta_h \kappa_h (P+I_a) S_e}{(1+\kappa_h I_h)^2} + \frac{\beta_h \kappa_h (P+I_a) S_u}{(1+\kappa_h I_h)^2}, y_2 = \frac{(1-\tau)\beta_h}{(1+\kappa_h I_h)} S_e + \frac{\beta_h}{(1+\kappa_h I_h)} S_u$$

Therefore;

$$\hat{G}(Y,Z) = \begin{bmatrix} \frac{(1-\tau)\beta_h(P+I_a)\left(\frac{\Lambda_h}{\mu_h} - S_e\right)}{(1+\kappa_h I_h)} + \frac{\beta_h(P+I_a)\left(\frac{\Lambda_h}{\mu_h} - S_u\right)}{(1+\kappa_h I_h)} \\ 0 \\ 0 \\ 0 \\ \frac{\beta_h P\left(\frac{\Lambda_a}{\mu_a} - S_a\right)}{1+\kappa_a I_a} \\ 0 \end{bmatrix} \quad 29)$$

Clearly, since S_e, S_u, S_a are bounded above by $\frac{\Lambda_h}{\mu_h}$ and $\frac{\Lambda_a}{\mu_a}$ respectively, it implies that $\frac{\Lambda_h}{\mu_h} \geq (S_e, S_u)$ and $\frac{\Lambda_a}{\mu_a} \geq S_a$. Thus, $\hat{G}(Y,Z) \geq 0, \forall Y, Z \in \Omega$. This concludes the proof that anthrax-free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

Numerical Simulation

To illustrate the theoretical results, numerical simulations were carried out. Model variables and parameters value for the numerical simulations source are listed in Table 3 below. Whenever parameter values were not available in the literature, we assumed realistic values for the purpose of illustration.

Table 3: Parameter values

Parameter	Value	Source
$\Lambda_h(\Lambda_a)$	$0.92(0.99)$	Osman *et al.*, (2018)
$\mu_h(\mu_a)$	$0.0001(0.0001)$	Osman *et al.*, (2018)
$\beta_h(\beta_a)$	$0.0001(0.02)$	Baloba and Seidu (2022)
τ	0.000017	Assumed
$C_h(C_a)$	$0.2(0.45)$	Baloba and Seidu (2022)
θ_h	0.005	Assumed
α_a	0.004	Osman *et al.*, (2018)
$\varepsilon_h(\varepsilon_a)$	$0.002(0.002)$	Assumed
ξ_a	0.45	Baloba and Seidu (2022)
η	0.8	Baloba and Seidu (2022)
$\kappa_h(\kappa_a)$	$0.6(0.6)$	Baloba and Seidu (2022)
p	0.5	Baloba and Seidu (2022)
γ_a	0.6	Osman *et al.*, (2018)
ϕ_h	0.04	Osman *et al.*, (2018)
$\delta_h(\delta_a)$	0.04	Osman *et al.*, (2018)

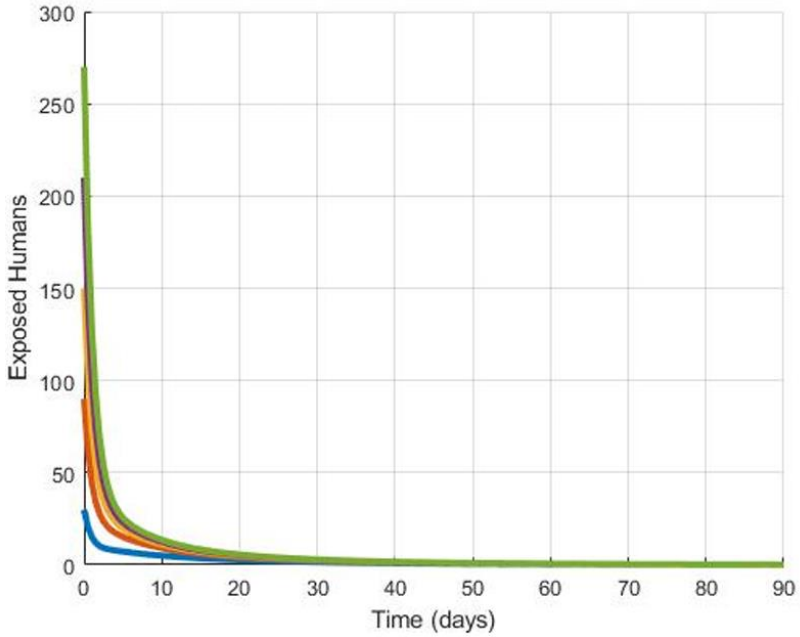

Figure 2: Time series plot of anthrax infection model for exposed human population with different initial conditions for $R_0 < 1$ with parameter values in table 4.1

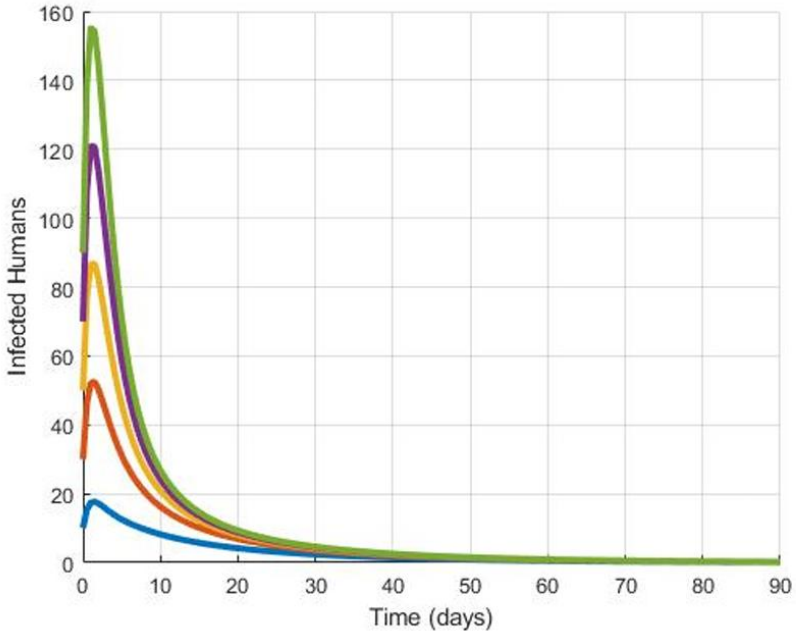

Figure 3: Time series plot of anthrax infection model for infected human population with different initial conditions for $R_0 < 1$ with parameter values in table 4.1

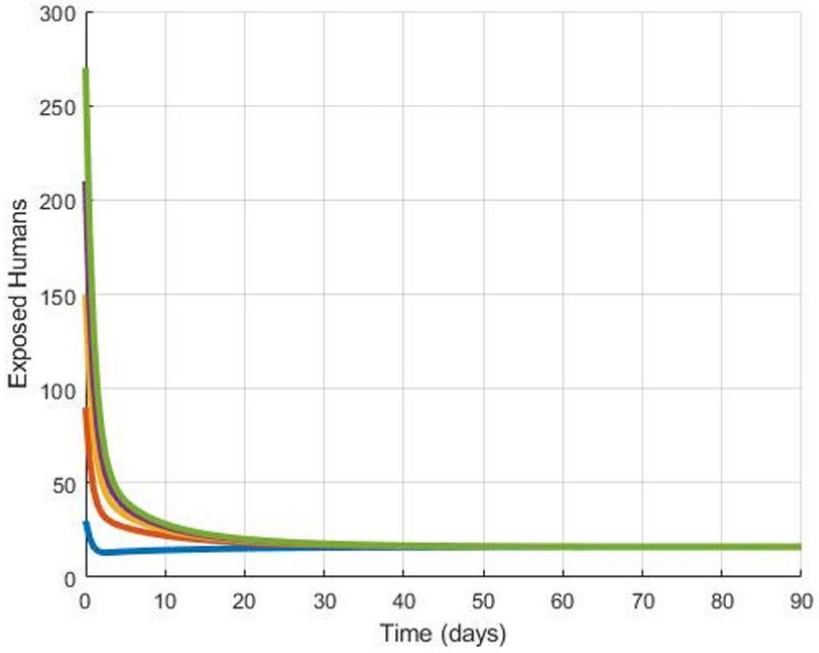

Figure 4: Time series plot of anthrax infection model for exposed human population with different initial conditions for $R_0 > 1$ with parameter values in table 4.1

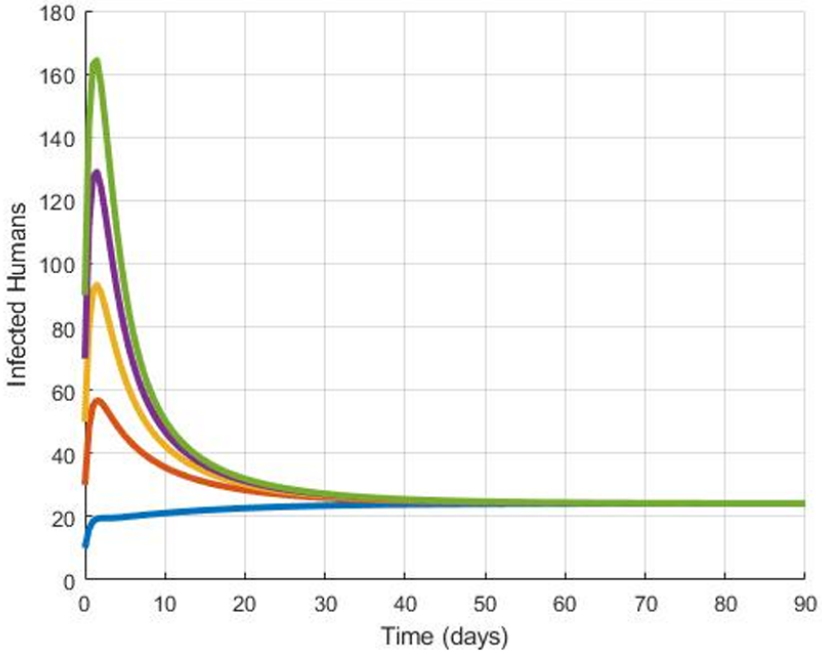

Figure 5: Time series plot of anthrax infection model for infected human population with different initial conditions for $R_0 > 1$ with parameter values in table 4.1

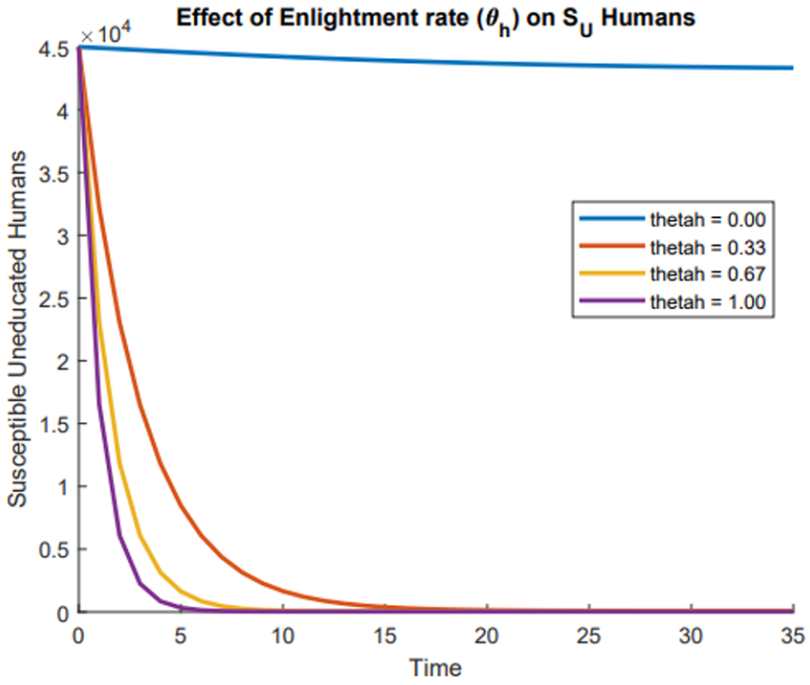

Figure 6: The effect of public health campaign on un educated susceptible humans with parameter values in table 4.1

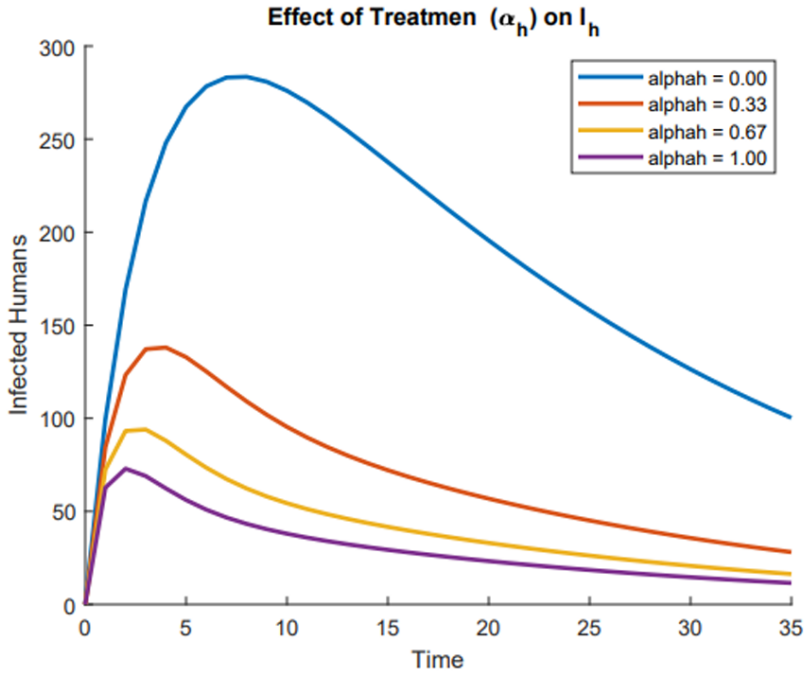

Figure 7: The impact of treatment on infected humans with parameter values in table 4.1

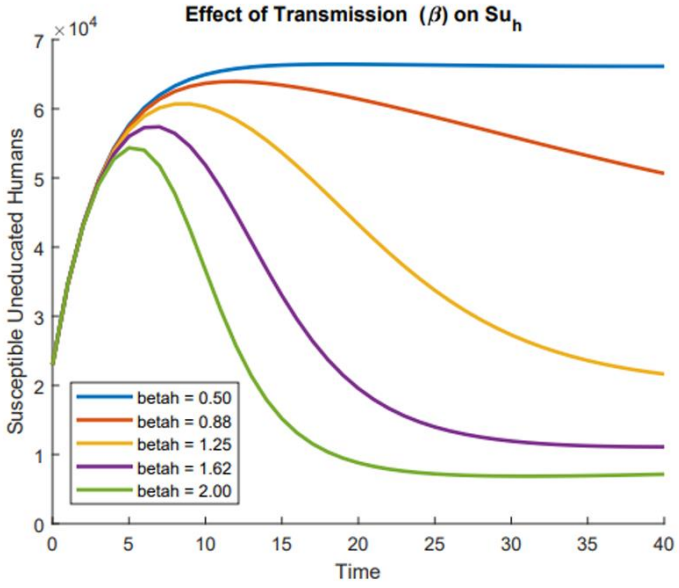

Figure 8: The effect of transmission rate β_h on un educated susceptible humans with parameter values in table 4.1

Discussion

Figure 2 to 3 shows that when $R_0 < 1$ all trajectories of exposed and infected humans converges to zero, for different values of initial conditions, which indicates the local stability for disease free equilibrium E_0, in human populations. Also using the values in table 4.1 varying the initial conditions, figure 4 and 5, shows that when $R_0 > 1$ the anthrax free equilibrium becomes unstable in human population.

Figure (6) Simulation results for the impacts of awareness program on the transmission dynamics of anthrax. The simulation assessed the influence of the awareness program on the un educated population by changing the level of awareness from zero to 33%, 67%, and 100%. The figure illustrates that lack of awareness leads to a high number of infected individuals, whereas 33% of awareness result in a slight decline. Furthermore, with a 67% level of awareness program, the number of infected individuals decreases significantly; however, the greatest reduction in infected individuals is observed with a 100% level of awareness program. Thus, this simulation finding is consistent with previous mathematical models in the literature that propose the implementation of public health awareness programs to prevent the spread of anthrax in a population.

Figure (7) shows the impact of treatment on infected human population at different rate. By increasing treatment rate ($\alpha = 0.33; 0.67; 1.00$). The disease can be eradicated in shortest possible time. This result clearly revealed that providing treatment for the infected individuals is crucial in saving the lives of those that are infected. Hence, the addition of treatment class has provided more insights into the control of anthrax in a given population. When illustrated in Figure 8, the susceptible uneducated persons decrease when anthrax infection rates increase. Humans decrease as they contract anthrax from the environment, eat meat from infected animals, or come into contact with pathogens in the environment.

Figure 8 shows that anthrax infections appear to be critical as years 47 goes by. Thus, this simulation result is in line with the existing mathematical models in the literature that an increase in the force of infection leads to a corresponding increase in the level of pathogens in the environment as well as an increase in the number of infective population

CONCLUSION

This study developed and analyzed a deterministic compartmental model to investigate the transmission dynamics of anthrax in human and animal populations while incorporating vaccination, treatment, and public health education as control measures. The mathematical analysis confirmed that the model is both mathematically and biologically well-posed, with all solutions remaining positive and bounded for all time. The basic reproduction number, R_0, derived via the next-generation matrix approach, was shown to play a crucial role in determining disease persistence. Specifically, the disease-free equilibrium was found to be locally and globally asymptotically stable when $R_0 < 1$, while a unique endemic equilibrium exists and is stable when $R_0 > 1$.Numerical simulations demonstrated that vaccination of animals, treatment of infected humans, and public health education are effective in reducing anthrax transmission. The results suggest that integrating these strategies can significantly mitigate outbreaks and safeguard both human and animal populations.

ACKNOWLEDGEMENT
We gratefully acknowledge the Department of Mathematics, Modibbo Adama University, Yola, for providing the necessary resources and a conducive environment that supported the successful completion of this research.

REFERENCES
Ahmed, B., Sultan, Y., Fatema, M.S.D., Ara, K., Begun, N., & Mostanzid, S.M. (2010) Anthrax: An emerging zoonotic disease in Bangladesh. Bangladesh Journal of Medical Microbiology, 4:46-50.

Awoonor-Williams, J.K., Apanga, P.A., Anyawie, M., Abachie, T., Boidoitsiah, & S., Opare, J.L. (2016). Anthrax outbreak investigation among humans and animals in Northern Ghana: Case Report.

Baloba, E.B., & Seidu, B. (2022) A mathematical model of anthrax epidemic with behavioural change. Mathematical modelling and control, 2(4): 253-256.

Castillo-Chavez, C., and Song B. (2004). Dynamical model of tuberculosis and their applications, *Math. Biosci. Eng.*, 1 (2), 361-404.

Decker, J. (2003) Deadly diseases and epidemics Anthrax. USA: Chelsea House Publishers.

Dragon, D.C., & Rennie, R.P. (1995) The ecology of anthrax spores, Tough but not invincible. The Canadian Veterinary Journal 36: 295–301.

Efraim, J.E., Irunde, J.I., & Kuznetsov, D. (2018). Modeling the transmission dynamics of anthrax disease in cattle and humans. J. Math. Comput. Sci. 8(6): 654-672.

Elijah, B.B., Baba,S. & Christopher, S.B. (2020) Mathematical analysis of the effects of controls on the transmission dynamics of anthrax in both animal and human populations, Computational and Mathematical Methods in Medicine, 1581358. https://doi.org/10.1155/2020/1581358.

Fasanella, A., Adone R., & Hugh-Jones M. (2016) Classification and management of animal anthrax outbreaks based on the source of infection. Ann Ist Super Sanita 50: 192-195.

Friedman, A., & Yakubu, A. (2013). Anthrax epizootic and migration: persistence or extinction. Mathematical Biosciences. 241(1): 137–144.

Hahn, B.D., & Furniss, P.R. (1983). “A deterministic model of an anthrax epizootic: threshold results,” Ecological Modelling. 20 (2): 233–241.

Hethcote, H. W. (2000) The Mathematics of infectious diseases. SIAM Rev. 42: 599–653. https://doi.org/10.1137/S0036144500371907.

Nigeria Centre for Disease Control (NCDC), (2023). Anthrax. Viewed June 27, 2023 from http://www.ncdc.gov.ng.

Samad, M.A. (2013). Public health threat caused by zoonotic diseases in Bangladesh. Bangladesh Journal of Veterinary Medicine. 95—120.

Shaibu, O., Oluwole, D.M. & David, M.T (2018). Mathematical Modelling of the Trans mission Dynamics of Anthrax in Human and Animal Population, Mathematical Theory and Modeling 8(6).

Sinkie, Z.M. & Murthy, S.N., (2016). Modeling and simulation study of anthrax attack on environment. Journal of Multidisciplinary Engineering Science and Technol ogy. 3(4): 4574-4578.

Stoltenow, C.L., (2021). Anthrax. NDSU Extension, USA.

Suma, A.P., Suresh, K.P., Gajendragad, M.R. & Kavya, B. A. (2017). Forecasting an thrax in livestock in Karnataka State using remote sensing and climatic variables. International Journal of Science and Research. 6: 1891–1897.

Van den Driessche, P., and James, W. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical *Biosciences*, 180, 29-48.

Yadeta, W., Giro, A., Amajo, M. & Jilo, K. (2020). Recent understanding of the epi demiology of animal and human anthrax in Ethiopia with emphasis on diagnosis, control and prevention interventions-review. World Jonal of Medical Science. 17: 1-9.