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Fractional order model of dynamical behavior and qualitative analysis of Anthrax with infected vector and saturation

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Abstract

We present a fractional order model to capture the transmission dynamics of anthrax infection with a nonlinear force of infection and its long-term impacts. An analysis with the standard Caputo–Fabrizio approach established that the model is well posed, and for the investigation on the stability of the model, an anthrax-free equilibrium as connected to the basic reproduction number $R_0 < 1$ and other important conditions that consolidate epidemiological feasibility of the model were established. Using a normalized forward sensitivity index, we conducted the sensitivity analysis of important variables, the contact rates, and the recruitment rate of the vectors to examine the effects of their variation on the dynamics of anthrax. These allow us to identify the most sensitive parameters that healthcare professionals need to pay attention to. The results from simulations also demonstrated that the presence of saturation instantly causes the system to approach an anthrax-free equilibrium (DFE), and our findings from the qualitative analysis compel us to recommend maximum hygiene practices for humans and domestic animals, early intervention, and the treatment of anthrax with a vaccine (as well as all other medical interventions) on the infected and periodic evaluation of these practices in households and the community at large to achieve a disease-free community.

Keywords and phrases: fractional model; incidence rate; anthrax; stability; sensitivity

1 Introduction

Bacillus anthracis is the bacteria that causes the infectious disease called anthrax. Both domestic and wild animals are affected by the disease. When vulnerable individuals come into touch with diseased animals or eat contaminated dairy and animal products, they can contract the anthrax virus. A zoonotic disease called anthrax is naturally occurring in soil and affects populations of humans and animals globally. People can become sick from tainted animal products or by having close contact with animals [1 - 4]. Infected animals, especially livestock, may die suddenly and unexpectedly from it, and it can occasionally be dangerous to people. Anthrax may generally be spread to animals in two ways: either by consuming a significant amount of spores found in soil or on plants or by stable insects called *Stomoxys calcitrans*, which feed on contaminated blood that leaks from the remains of animals that

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have contracted the disease [5 - 6]. Applying immunizations before exposure is the only effective way to control anthrax and is advisable to do so between 2–4 weeks prior to the anticipated endemic season [5]. Anthrax spores may survive for up to two centuries because of their remarkable resilience to temperature fluctuations, desiccation, heat, and a range of chemical agents [7]. Furthermore, in cattle, anthrax infections frequently worsen without showing any signs (symptoms) [5]. The time frame in such a scenario could be critical for averting anthrax outbreaks.

In addition to herbivorous animals, predators that eat the contaminated carcasses of anthrax-infected prey are also at risk from anthrax. However, compared to herbivores, carnivores often have lower infection and death rates. According to the World Health Organization (WHO), the World Anthrax Data Site has recorded anthrax outbreaks in animals from around 200 nations, especially in areas with large populations of both humans and animals. For example, the exact reason of the anthrax deaths of a variety of animals in Etosha National Park, including ostriches and elephants, was still unclear. Three captive cheetahs were reported dead in November 2004 at the Jwana Game Reserve in Jwanen, Botswana. Meat from deceased red hartebeest infected with anthrax had been given to them [8 -9]. Africa, the Middle East, and Asia have relatively high rates of anthrax infection in wild animals and livestock, with periodic outbreaks and epizootics [10]. Anthrax is quite widespread in Bangladesh and India, two nations in southern Asia; outbreaks and diseases in both humans and animals are often reported [11 -12]. If treatment measures are not taken, the most common cutaneous type of anthrax infection can have a morbidity (fatality) rate of up to 20%; however, with appropriate treatment, it can drop to less than 1% [13].

Both natural phenomena and the dynamics of infectious disease transmission may be explained by mathematical models. In epidemiology, these models are essential for controlling infectious diseases. These models are able to clearly describe the dynamics and mechanisms of disease transmission [14 - 15]. Lotka and Volterra introduced the field of prey–predator ecosystem in mathematical research, which was termed the Lotka–Volterra model [16 - 17]. In mathematical modeling, Kermack and Kendrick coined the term “epidemiology” and investigated an SEIRS compartment model [18 - 19]. For a while now, there have been proposals and research done on complex models for the transmission dynamics of diseases, including periodic orbits, Hoff bifurcations, and multiple equilibria. They provide a clear qualitative depiction of the dynamics of the disease, together with improved analysis and consequences for disease forecasting [20 - 21]. The authors of [1] examined how a continuous vaccine affected an anthrax model, but they never took into account the consequences of the best possible control. Based on their studies, theoretical findings under continuous vaccination, the disease model’s transmission resembles dynamics in the absence of vaccination. Additionally, [22] created a model by taking the influence of vaccines into account and came to a conclusion for the ideal vaccination coverage threshold needed for disease elimination. On the other hand, [23] employed optimum control while examining a nonlinear SIR epidemic model that included a vaccination plan.

No nation can really declare itself free of anthrax since the disease’s spores may survive for a very long period in the soil. Nonetheless, anthrax epidemics tend to occur more often in developing nations.

Furniss and Hahn conducted groundbreaking research on anthrax modeling from a mathematical modeling standpoint when they examined an anthrax epizootic in South Africa's Kruger National Park [24]. The same researchers put up a different deterministic model with some threshold findings in 1983 [25]. Friedman and Yakubu added elements such as migration, growth, and the host's natural death [26] to the model put forward in [24], adding a predetermined time delay. Mushayabasa suggested a fresh approach that was based on the model of Hahn and Furniss [25]. The function of human labor (as a control mechanism) in cleaning up contaminated carcasses, including the soil or surrounding region, is also included in their model [27]. Two distinct models investigating the spread and management of anthrax infection within two frameworks were provided in [28]. They investigated the effects of the incubation time and carcass clearance in the first framework. They looked at how the vector population affected the transmission and management of anthrax in the second approach. Using vaccination as a control, Kimathi and Wainaina [29] looked at the dynamics of anthrax spread in animal populations. The co-dynamics of anthrax and listeriosis infections were investigated, and the qualitative and quantitative link between them within preventative and treatment strategies was researched in [30]. According to their research, anthrax infection may raise the chance of listeriosis, but the listeriosis disease itself is not linked to an increased risk of anthrax. Their results show that while listeriosis infection is not linked to an increased risk of anthrax infection, anthrax infection is associated with a higher likelihood of listeriosis infection. A novel fractional ordered model in the sense of the Caputo–Fabrizio fractional derivative was suggested in [31]. This work was inspired by the work of [29]. They examined the asymptotic stability of the equilibrium states that are endemic and disease-free in order to calculate the fundamental reproduction number R_0 of the fractional system.

Researchers' interest in disease modeling, particularly the modeling of diseases such as HIV and cancer, has increased recently. The SARS-CoV-2 virus is the source of the COVID-19 pandemic, which has lately garnered a lot of attention, with over 21 million confirmed cases and 758,000 fatalities recorded. During the COVID-19 epidemic, mathematical models were essential in predicting different waves, and research in this field is still ongoing [4, 32].

Our examination of the literature indicates that, compared to diseases such as cancer or HIV, modeling the anthrax disease has received comparatively less attention from researchers. The ongoing threat that anthrax poses in developing nations (Africans and the likes) is a strong incentive for us to investigate the mathematical modeling of anthrax in this research work.

1.1 Epidemiology of Anthrax in Nigeria

Following the discovery of widespread death resulting from the pathognomonic signs of the disease in a multi-species farm in Gajiri Village, Suleja, Niger State, Nigeria's index anthrax case was confirmed by the Nigerian Center for Disease Control (NCDC). About 100 cows (49 foreign and 51 native varieties), 73 sheep (a breed of fat-tailed sheep from Sudan), and 19 goats were present on the property where the index case was located. On 27 June 2023, the circumstances that led to the verified anthrax epidemic began. Thirteen cattle and ten sheep were among the

thirty-five instances (twenty cattle and fifteen sheep) that had been reported as of 13 July 2023, the day the samples were collected. The national reference laboratory made a confirmation diagnosis of anthrax on 16 July 2023 (Nigerian Veterinary Research Institute). In VOM, Plateau State, although animals are driven to graze on natural grass, the majority of the cows were grown in large production systems. On 19 July 2023, the global disease notice was transmitted to the World Animal Health Information system, and an urgent notification was issued to the World Organization for Animal Health on 17 July 2023.

In addition, more research is being done to determine the infection's origin and how it spread to other farms and people. Although reports of human exposure exist, no human symptoms or deaths have been documented yet. To identify, isolate, and monitor all affected individuals, the FMARD and the Nigeria Centre for Disease Control and Prevention (NCDC) are presently collaborating closely with the Ministries of Agriculture and Health of Niger State. The incident manager has been designated by the NCDC to ensure efficient response coordination, and the incident management system (IMS) has been activated at level two (2). The probability of an epidemic and its possible effects on humans were rated as HIGH in the risk assessment carried out by the human health sector (NCDC).

1.2 Treatment of Anthrax

Adequate immunization of vulnerable vector populations lowers the likelihood of it spreading to humans because the disease is zoonotic and preventable with the recommended vaccine. Therefore, minimizing contact with diseased vectors and managing vectors with anthrax effectively removes the danger to humans. Annual preventative vaccination is the main method of controlling animal anthrax, but if an epidemic starts, other methods of controlling the disease might also be effective, including ring vaccination, the correct disposal of carcasses, and the separation of newly acquired or infected vectors (animals) [33]. For vectors in the red zone as well as other vectors around the nation, ring vaccination is recommended. Research has indicated that immunization with either the non-living anthrax vaccine prototypes or the Sterne live anthrax spore vaccine, which is a non-pathogenic, non-encapsulated strain of *Bacillus anthracis*, has produced an immune response to the disease [34]. Health commissions must warn that simultaneous antibiotic treatment might significantly reduce the efficiency of the live-attenuated vaccine because animal producers, out of fear, may give antibiotics to animals in order to avoid anthrax. Therefore, field personnel need to make sure that animals on antibiotic programs are vaccinated again [33, 35]. Another crucial element of prevention in humans is the prescription of antibiotics as post-exposure interventions. Post-exposure interventions should be administered to danger groups, such as veterinarians, animal health professionals, and anyone engaged in the removal of carcasses, and personal protective equipment (PPE) must be worn [36].

1.3. Forces of Infections and Incidences

Various researchers have utilized some incidence functions, which include the bilinear incidence rate βSI , where β is a positive constant S is the susceptible individual in the population and I is infected people in the population.

Similarly, some researcher's also make use of $\frac{\beta SI}{1+PS}$ and $\frac{\beta SI}{1+PI}$, where $p>0$ is the saturated incidence rate.

Most studies employ the bilinear rate of incidence βSI and the conventional rates $\beta SI/N$; there has been less focus on applying the incident rate of saturation in epidemiological models. This served as inspiration for this study.

1.4. The Sensitivity Analysis Theory

Because anthrax is so widespread, it is imperative that control measures be designed, assessed, and put into action in order to eradicate the virus's ever-present nature. In this instance, this means testing and examining the dynamics of the disease to identify the sensitive parameters causing the outbreak; this requires performing a sensitive analysis on the behavioral changes of the disease's transmission.

An analysis of the sensitivity examines how changes in the inputs might affect a model's outputs. A sensitivity analysis basically identifies the parameters and starting circumstances (inputs) that have the greatest impact on the model's outputs or quantities of interest. This study is significant in the first place because it indicates to the scientist (researchers) which parameters require the greatest amount of arithmetical consideration [34-35].

1.4. Preliminary and Definitions

This section includes some notations, definitions, and established results that are required for the sequel. This study uses Liouville–Caputo's fractional derivative.

Definition 1. Let U be a real function in the range C_χ , where $\chi \in \mathbb{R}, t > 0, \exists$ a $n > \chi$, which is real, then

$$U(t) = t^n k(t), \tag{1}$$

and for $k \in C[0, \infty]$, it is said to be in the range C_χ^m if and only if $U^{(m)} \in C_\chi, m \in \mathbb{M}$.

Definition 2. The Transformation of Laplace is given by

$$F(s) = L\{F(t)\}(s) = \int_0^{\infty} e^{-st} F(t) dt, \quad (2)$$

where $F(t)$ is a valued function of t .

Definition 3. The fractional integrating operator of order $\alpha \geq 0$ of the Riemann Liouville of function $U \in L^1(a, b)$ is defined as

$$I^\alpha U(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} U(\tau) d\tau, \quad t > 0, \alpha > 0 \quad (3)$$

where Γ is the function of gamma and $I^0 U(t) = f(t)$.

Definition 4. The fractional derivative in the sense of Liouville–Caputo is given by [35, 39].

$$D^\alpha U(t) = I^{n-\alpha} D^n U(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-\tau)^{n-\alpha-1} U^{(n)}(\tau) d\tau, \quad (4)$$

Where $n \in \mathbb{N}$ and $n-1 < \alpha \leq n$ for $t > 0$.

Definition 5. The Mittag–Leffler generalized function, also known as two parameter Mittag–Leffler function, is given by

$$E_{\alpha, \beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}, \quad \operatorname{Re}(\alpha) > 0, \beta, z \in \mathbb{C} \quad (5)$$

When $\beta = 1$, the Mittag–Leffler function with one parameter is derived as a particular instance of this function and is denoted as

$$E_{\alpha, \beta}(Z) = E_{\alpha, 1}(z) = \sum_{k=0}^{\infty} \frac{Z^k}{\Gamma(\alpha k + 1)}, \quad \operatorname{Re}(\alpha) > 0 \text{ for } Z \in \mathbb{C} \quad (6)$$

α and β can as well take any form of different values in order to show that the Mittag–Leffler function can represent important special cases.

Lemma 1. [36]. For $\mu > 0$ and $0 < \alpha \leq 1$, the function $E_{\alpha, \beta}(-\mu z^\alpha)$, $\alpha \leq \beta$ is monotonically decreasing positive function of $z > 0$.

Definition 6. For the Caputo’s derivative, its Laplace transform is given by

$$L\{D^\alpha N(t)(s)\} = S^\alpha N(s) + \sum_{k=0}^{n-1} S^{n-k-1} N^k(0), \quad \operatorname{Re}(\alpha) > 0, \quad n-1 < \alpha \leq n. \quad (7)$$

Lemma 2. Putting into consideration the two parameters of Mittag–Leffler function, we then have the Laplace transform formula as

$$L\{x^{\beta-1} E_{\alpha,\beta}(\mu^* t^\alpha)\}(s) = \frac{s^{\alpha-\beta}}{s^\alpha - \mu^*}, \quad \operatorname{Re}(z) > 0 \quad (8)$$

where $\alpha, \beta, \mu^* \in \mathbb{C}$, $\operatorname{Re}(\beta) > 0$, $\operatorname{Re}(\alpha) > 0$

Lemma 3. Let $\Psi \in C[a, b]$ and $D^\alpha \Psi \in (0, b]$, and let $0 < \alpha \leq 1$; therefore, we have

$$U(t) = U(a) + \frac{1}{\Gamma(\alpha)} D^\alpha U(\chi)(t-a)^\alpha \quad \text{for } 0 \leq \chi \leq t \text{ and for } t \in (a, b].$$

The above lemma is mean value theorem, which is the generalized one.

Lemma 4. For $U \in C[a, b]$ and $D^\alpha U \in (a, b]$, let $0 < \alpha \leq 1$; hence, we have the following:

(a) For $D^\alpha U(t) \geq 0$, $\forall t \in (0, b)$, the function u is increasing.

(b) For $D^\alpha U(t) \leq 0$, $\forall t \in (0, b)$, the function u is decreasing.

2 Model formulation

According to the proposed model, the whole system (population) is represented by the letter “N” and has been split into two primary parts: “N_v” and “N_h” for vectors and humans, respectively. The susceptible, exposed, infected, and recovered human sub-populations (S_h, E_h, I_h, and R_h) are present in 'N_h', respectively, from this subdivision; similarly, for the vector sub-population, N_v, we have susceptible, infected, and recovered (S_v, I_v, and R_v), assuming that the domestic animals can recover from the disease with appropriate control techniques.

Additionally, the recruitment/immigration of humans and vectors into the population in question occurs through birth or immigration at rates of Λ_h and Λ_v , respectively. Humans and vectors contract the anthrax virus through contact with diseased animals or the consumption of tainted dairy and animal products containing *Bacillus anthracis* at forced or saturated rates of infection, Φ_1 and Φ_2 .

$$\Phi_1 = \frac{\beta_1 I_H}{1 + a_H I_H} + \frac{\beta_2 I_v}{1 + a_v I_v} \quad (8)$$

$$\Phi_2 = \frac{\beta_3 I_v}{1 + a_v I_v} \quad (9)$$

where a_H is the saturation factor for humans and a_v is the saturation force for vectors.

The effective contact rates that can result in infection and saturation factors are represented by $\beta_1, \beta_2, \beta_3$, and a_H, a_v , respectively.

The results of this study show that the saturation rates and infection forces in (9) and (10) above predict the behavioral change in the model when I_h and I_v grow very big in the system as well as their repressive effects when simulated. This is the reason this model has taken into account the specific saturation incidence rate.

In this model, we have also taken into account the possibility that individuals may lose their immunity, making them susceptible to infection again at the rate ϱ after receiving treatment, with a recovery rate of η . However, in severe cases of anthrax sickness, people may pass away at a rate of δ because of the virus's virulence.

2.1 The Anthrax model's equations

The set of time fractional differential equations designed to describe the dynamics of the disease is basically on the descriptions and assumptions of the above model (Figure 1).

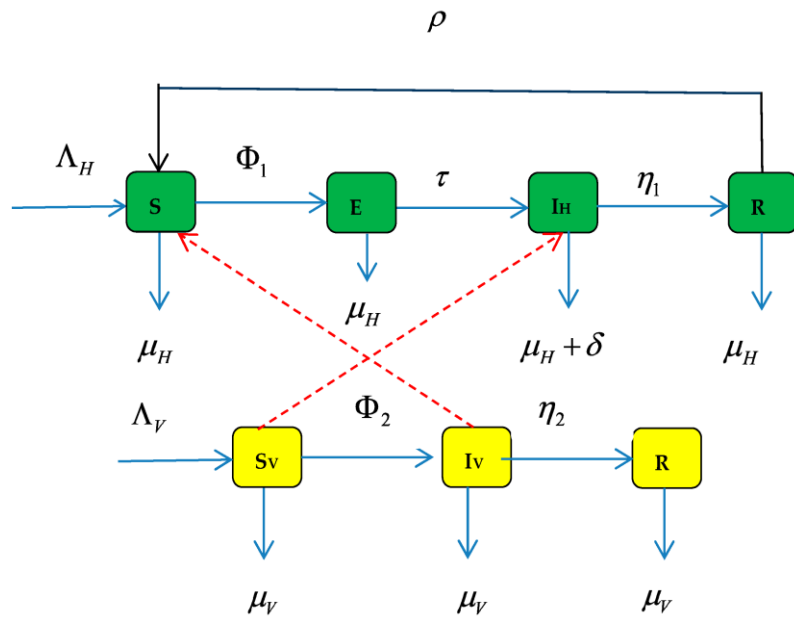


Figure 1. Schematic flow of the anthrax model.

$$\left. \begin{aligned}
 D^\alpha S_H(t) &= \Lambda_H - \mu_H S_H - \Phi_1 S_H + \rho R_H \\
 D^\alpha E_H(t) &= \Phi_1 S_H - \mu_H E_H - \tau E_H \\
 D^\alpha I_H(t) &= \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\
 D^\alpha R_H(t) &= \eta_1 I_H - \mu_H R_H - \rho R_H \\
 D^\alpha S_v(t) &= \Lambda_v - \mu_v S_v - \Phi_2 S_v \\
 D^\alpha I_v(t) &= \Phi_2 S_v - \mu_v I_v - \eta_2 I_v \\
 D^\alpha R_v(t) &= \eta_2 I_v - \mu_v R_v
 \end{aligned} \right\} \quad (10)$$

Here $D^\alpha = \frac{d^\alpha}{dt^\alpha}$, for $0 < a \leq 1$ in the sense of the Caputo fractional derivative of order a

Table 1. Parameters and Descriptions

Parameters	Description
S_H	Human Susceptible
E_H	Human Exposed
I_H	Human Infected
R_H	Human Recover
S_V	Vector Susceptible
I_V	Infected vector
R_V	Recovered vector
Λ_H	Immigration/recruitment rate for human
Λ_V	Immigration/recruitment rate for vector
Φ_1	Force of infection for human
Φ_2	Force of infection for vector
ρ	Loss of immunity human
μ_H	Natural death rate for human
μ_V	Natural death rate for vector
τ	Rate of Progression from exposed to infected class of human
η_1	Rate of Progression from infected to recovery class of human
η_2	Rate of Progression from infected to recovery class of vector
β_1	The rate of effective contact between for humans
β_2	Rate of effective contact between domestic vector and <i>Bacillus anthracis</i>
β_3	Rate of contact leading to infection for the vector

δ	Disease-induced death
a_H	Saturation factor for human
a_V	Saturation factor for vector

3 Analysis of the model

3.1 Existence and Uniqueness of the Model Solution

To demonstrate the uniqueness and existence of solutions to the system (11), Lemma 4 as it has been given above, is required, which is given as follows: For $U \in C[a, b]$ and $D^\alpha U \in (0, b]$, let $0 < a \leq 1$. Hence, we have the following:

(a) For $D^\alpha U(t) \geq 0$, $\forall t \in (0, b)$, the function u is increasing.

(b) For $D^\alpha U(t) \leq 0$, $\forall t \in (0, b)$, the function u is decreasing.

The proof of the above lemma is a direct consequence of Lemma 3, which is the generalized mean value theorem.

3.2. Boundedness and Positivity of the Model

In this section, we verify and ascertain if our proposed model is epidemically and mathematically accurate. Moreover, each compartment's solutions are non-negative (positive). The whole vector population is represented by $N_V = S_V + I_V + R_V$, while the total human system is represented by $N_H = S_H + E_H + I_H + R_H$.

Therefore,

$$N_H = S_H + E_H + I_H + R_H$$

$$N_V = S_V + I_V + R_V$$

which yields

$$\left. \begin{aligned} D^\alpha N_H(t) &= D^\alpha S_H(t) + D^\alpha E_H(t) + D^\alpha I_H(t) + D^\alpha R_H(t) \\ D^\alpha N_V(t) &= D^\alpha S_V(t) + D^\alpha I_V(t) + D^\alpha R_V(t) \end{aligned} \right\} \quad (11)$$

From (12), we can conclude that for both the human and vector compartment, we have

$$\left. \begin{aligned} D^\alpha N_H(t) &= \Lambda_H - \mu_H N_H \\ D^\alpha N_V(t) &= \Lambda_V - \mu_V N_V \end{aligned} \right\} \quad (12)$$

with $\delta = 0$ for both the human and vector compartment, i.e., the disease-induced death in order to achieve (13).

Theorem 1. Let (S_H, E_H, I_H, R_H) and (S_V, I_V, R_V) be the solution to the anthrax fractional model equation (11) in an epidemiologically viable area Γ , where

$$\left. \begin{aligned} \Gamma &= (S_H, E_H, I_H, R_H) \in R_+^4 : N_H \leq \frac{\Lambda_H}{\mu_H} \\ \Gamma &= (S_V, I_V, R_V) \in R_+^3 : N_V \leq \frac{\Lambda_V}{\mu_V} \end{aligned} \right\} \quad (13)$$

Proof. With the Laplace transform applied on both sides of (13) (for both humans and vectors) and using the Definition 6, we have

$$\left. \begin{aligned} S^\alpha N_H(s) + S^{\alpha-1} N(0) &= \frac{\Lambda_H}{s} - \mu_H N_H(s) \\ S^\alpha N_V(s) + S^{\alpha-1} N(0) &= \frac{\Lambda_V}{s} - \mu_V N_V(s) \end{aligned} \right\} \quad (14)$$

After little modifications and rearrangement on (15), we have

$$\left. \begin{aligned} N_H(s) &= \frac{\Lambda_H}{s(s^\alpha + \mu_H)} + \frac{s^{\alpha-1}}{s^\alpha + \mu_H} N(0) \\ N_V(s) &= \frac{\Lambda_V}{s(s^\alpha + \mu_V)} + \frac{s^{\alpha-1}}{s^\alpha + \mu_V} N(0) \end{aligned} \right\} \quad (15)$$

After considerable simplification and application of the Laplace inverse transform and (Lemma 2), as given earlier on (16), the following was derived:

$$\left. \begin{aligned} N_H(t) &= N(0)E_\alpha(-\mu_H t^\alpha) + \frac{\Lambda_H}{\mu_H} [1 - E_\alpha(-\mu_H t^\alpha)] \\ N_V(t) &= N(0)E_\alpha(-\mu_V t^\alpha) + \frac{\Lambda_V}{\mu_V} [1 - E_\alpha(-\mu_V t^\alpha)] \end{aligned} \right\} \quad (16)$$

Applying (17) with Lemma 1, as t tends to ∞ , the following are obtained:

$$\left. \begin{aligned} N_H(t) &\leq \frac{\Lambda_H}{\mu_H} \\ N_V(t) &\leq \frac{\Lambda_V}{\mu_V} \end{aligned} \right\} \quad (17)$$

Thus, every solution for system (11) is included inside the set Γ ; hence, Γ is bounded for both the human and vector population.

Hence, from Equations (13)–(18), we have been able to verify that the solutions for the various compartments are bounded and that the model is well posed both mathematically and epidemiologically.

This ends the proof.

3.3 Positivity

For all time $t > 0$, the fractional order model's solution in system (11) with positive initial conditions will continue to be positive and non-negative.

Proof

Using the Mittag–Leffler function, also known as two parameter function (5) and (6), we derive

$$E_{\alpha,\beta} = \sum_{k=0}^{\infty} \frac{(Z)^k}{\Gamma \alpha k + \beta}$$

Replacing Z with our population $N_H(t)$ and $N_V(t)$, respectively and separately, where $k = 0, 1, 2, \dots$ and $\alpha = \beta = 1$, we derive

$$\left. \begin{aligned} E_{1,1} &= \sum_{k=0}^{\infty} \frac{(N_H(t))^k}{\Gamma \alpha k + \beta} = \frac{(N_H(t))^0}{\Gamma(0+1)} + \frac{(N_H(t))^1}{\Gamma(1+1)} + \dots \\ E_{1,1} &= \sum_{k=0}^{\infty} \frac{(N_V(t))^k}{\Gamma \alpha k + \beta} = \frac{(N_V(t))^0}{\Gamma(0+1)} + \frac{(N_V(t))^1}{\Gamma(1+1)} + \dots \end{aligned} \right\} \quad (18)$$

Neglecting the higher terms, we derive

$$\left. \begin{aligned} E_{1,1} &= \sum_{k=0}^{\infty} \frac{(N_H(t))^k}{\Gamma \alpha k + \beta} \approx \frac{1}{\Gamma(1)} + \frac{(N_H(t))^1}{\Gamma(2)} \\ E_{1,1} &= \sum_{k=0}^{\infty} \frac{(N_V(t))^k}{\Gamma \alpha k + \beta} \approx \frac{1}{\Gamma(1)} + \frac{(N_V(t))^1}{\Gamma(2)} \end{aligned} \right\} \quad (19)$$

$$\Gamma(1) = 1$$

$$\left. \begin{aligned} E_{1,1} &= \frac{(N_H(t))}{\Gamma \alpha k + \beta} \approx 1 \\ E_{1,1} &= \frac{(N_V(t))}{\Gamma \alpha k + \beta} \approx 1 \end{aligned} \right\} \quad (20)$$

We have also been able to verify that the model is positive for time (t) based on equations of model (11)

This ends the proof.

3.4. Presence of the disease-free equilibrium points

The term “disease-free system (equilibrium) (DFE) points” refers to stable-state solutions when the population as a whole becomes immune to anthrax infection.

We have

$$\left. \begin{aligned} S_H^0 \neq 0, S_V^0 \neq 0, \\ \text{for } \left. \begin{aligned} S_H^0 \neq 0, E_H^0 = 0, I_H^0 = 0, R_H^0 = 0 \\ S_V^0 \neq 0, I_H^0 = 0, R_H^0 = 0 \end{aligned} \right\} \end{aligned} \right\} \quad (21)$$

Hence, system (18) becomes

$$\left. \begin{aligned} \Lambda_H - \mu_H S_H^0 &= 0 \\ \Lambda_V - \mu_V S_V^0 &= 0 \end{aligned} \right\} \quad (22)$$

This yields

$$\left. \begin{aligned} S_H^0 &= \frac{\Lambda_H}{\mu_H} \\ S_V^0 &= \frac{\Lambda_V}{\mu_V} \end{aligned} \right\} \quad (23)$$

Therefore, the disease-free equilibrium point for both the human and vector population is

$$E^0 = (S_H^0, E_H^0, I_H^0, R_H^0, S_V^0, I_V^0, R_V^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0 \right) \quad (24)$$

Equation (25) is the DFE point for the system.

3.5. Computation of the R_0 (reproduction number) of the human–vector model

In an epidemic model, the R_0 is an essential concept. This figure indicates the typical number of later infections in the exposed section of the population among all individuals in a population that is fully susceptible and infected by an infectious person already in the infected compartment. Similarly, R_0 is a crucial metric that tells us whether or not a disease will spread over the population. The “next generation matrix method” will be used in order to calculate it.

Using system (11), the model’s fundamental reproduction number is obtained using $R_0 = \Delta(FV^{-1})$, where Δ is the dominant eigenvalue of FV^{-1} , which yields R_{HV} ; here, R_{HV} is the combination of both compartments (human and vector). V is the linear system of additional transfer terms with infection, and F is the linear system matrix of the new infection. Consequently, for simplicity, we calculate separately the human reproduction number, R_H , and vector reproduction number, R_V , and then obtain the product.

Proof. We implement the next generation matrix [41 - 44] on the human compartment, that is

$$\begin{aligned} D^\alpha S_H(t) &= \Lambda_H - \mu_H S_H - \Phi_1 S_H + \rho R_H \\ D^\alpha E_H(t) &= \Phi_1 S_H - \mu_H E_H - \tau E_H \\ D^\alpha I_H(t) &= \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\ D^\alpha R_H(t) &= \eta_1 I_H - \mu_H R_H - \rho R_H \end{aligned} \quad (25)$$

and $\Phi_1 = \left(\frac{\beta_1 I_h}{1 + a_h I_h} + \frac{\beta_2 I_v}{1 + a_v I_v} \right) S_H$; here, $a_H = a_V = \frac{\beta_2 I_V}{1 + a_V I_V} = 0$ since the assumption is that the infection is more dominant in the exposed and the infected class of the human population.

$$F_H = \begin{bmatrix} \Phi_1 S_h \\ 0 \end{bmatrix} = \begin{bmatrix} \frac{\partial F_1}{\partial E_H} & \frac{\partial F_1}{\partial I_H} \\ \frac{\partial F_2}{\partial E_H} & \frac{\partial F_2}{\partial I_H} \end{bmatrix} = \begin{bmatrix} 0 & \beta_1 S_H \\ 0 & 0 \end{bmatrix} \quad (26)$$

$$\text{Similarly, } V_H = \begin{bmatrix} -\mu_H E_H - \tau E_H \\ \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \end{bmatrix} = \begin{bmatrix} \frac{\partial V_1}{\partial E_H} & \frac{\partial V_1}{\partial I_H} \\ \frac{\partial V_2}{\partial E_H} & \frac{\partial V_2}{\partial I_H} \end{bmatrix} = \begin{bmatrix} -(\mu_H + \tau) & 0 \\ \tau & -(\mu_H + \delta + \eta_1) \end{bmatrix} \quad (27)$$

$$V_H^{-1} = \frac{\text{Adj}(V)}{|V|},$$

using

$$R_{0H} = \Delta(F_H V_H^{-1}).$$

We have

$$F_H V_H^{-1} = \begin{bmatrix} \frac{\tau \beta_1 S_H}{(\mu_H + \delta + \eta_1)(\mu_H + \tau)} & \frac{\beta_1 S_H}{(\mu_H + \delta + \eta_1)} \\ 0 & 0 \end{bmatrix}$$

$$|F_H V_H^{-1} - \lambda I| = \begin{vmatrix} \frac{\tau \beta_1 S_H}{(\mu_H + \delta + \eta_1)(\mu_H + \tau)} - \lambda & \frac{\beta_1 S_H}{(\mu_H + \delta + \eta_1)} \\ 0 & 0 - \lambda \end{vmatrix}$$

The most dominant eigenvalue was utilized, which is the spectral radius; therefore,

$$R_{0H} = \frac{\tau \beta_1 S_h}{(\mu_H + \delta + \eta_1)(\mu_H + \tau)}. \text{ Recall that } S_H \leq \frac{\Lambda_H}{\mu_H}; \text{ hence,}$$

$$R_{0H} = \frac{\tau \beta_1 \Lambda_H}{(\mu_H + \delta + \eta_1)(\mu_H + \tau) \mu_H} \quad (28)$$

It is obvious that the effective contact rate of the human population β_1 will contribute to the reproduction number in the human population.

In the same vein, we calculate the reproduction number for the vector population R_v

Considering the vector compartment,

$$\begin{aligned}
D^\alpha S_v(t) &= \Lambda_v - \mu_v S_v - \Phi_2 S_v \\
D^\alpha I_v(t) &= \Phi_2 S_v - \mu_v I_v - \eta_2 I_v \\
D^\alpha R_v(t) &= \eta_2 I_v - \mu_v R_v
\end{aligned} \tag{29}$$

and

$$\begin{aligned}
\Phi_2 &= \left(\frac{\beta_3 I_v}{1 + a_v I_v} \right) S_v. \text{ Note that } a_v = 0 \\
F_v &= \begin{bmatrix} \Phi_2 S_v \\ 0 \end{bmatrix} = \begin{bmatrix} \frac{\partial F_1}{\partial E_v} & \frac{\partial F_1}{\partial I_v} \\ \frac{\partial F_2}{\partial E_v} & \frac{\partial F_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} 0 & \beta_1 S_v \\ 0 & 0 \end{bmatrix}
\end{aligned} \tag{30}$$

$$V_v = -(\mu_v + \eta_2) I_v, \quad \frac{\partial V_v}{\partial I_v} = -(\mu_v + \eta_2)$$

Using

$$\begin{aligned}
R_{0v} &= \Delta(F_v V_v^{-1}) \text{ and } |F_v V_v^{-1} - \lambda I|, \\
R_{0v} &= \frac{\beta_3 S_v}{(\mu_v + \eta_2)}. \text{ Recall that } S_v \leq \frac{\Lambda_v}{\mu_v}; \text{ therefore,} \\
R_{0v} &= \frac{\beta_3 \Lambda_v}{(\mu_v + \eta_2) \mu_v}
\end{aligned} \tag{31}$$

It is also obvious that the infection rate of the vector population β_3 will influence the reproduction number in the vector population.

The product of the reproduction number for both human and vector compartments is given as

$$\begin{aligned}
R_{0HV} &= R_{0v} \times R_{0H} = R_{0HV}^2 \\
R_{0HV} &= \sqrt{\frac{\beta_1 \beta_3 \Lambda_v \Lambda_H \tau}{\mu_v \mu_H (\mu_v + \eta_2) (\mu_H + \delta + \eta_1) (\mu_H + \tau)}}
\end{aligned} \tag{32}$$

The possibility that a human will survive the exposed stage before becoming infectious is $\frac{\tau}{(\mu_H + \tau)}$.

Similarly, $\frac{1}{(\mu_v + \eta_2)}$ is the possibility that vector population might also endure the exposed stage before becoming infectious, where $\frac{1}{(\mu_H + \delta + \eta_1)}$ is the median duration of the infectious time in the human population.

This ends the proof. $\square$

3.6. Local stability analysis of Anthrax at (DFE)

After establishing the existence of a disease-free equilibrium for our proposed model, we will now conduct stability analyses on the system to infer its long-term effects regarding whether or not it is stable.

Theorem 2: If all the population's Jacobian eigenvalues are non-positive (negative) real values, then our system's DFE is locally asymptotically stable If $R_{HIV} < 1$.

Proof. In order to establish the theorem above, we calculate the Jacobian of the model's system (11) at its disease-free equilibrium (DFE) E^0 .

$$\left. \begin{aligned} F_1 &= \Lambda_H - \mu_H S_H - \Phi_1 S_H + \rho R_H \\ F_2 &= \Phi_1 S_H - \mu_H E_H - \tau E_H \\ F_3 &= \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\ F_4 &= \eta_1 I_H - \mu_H R_H - \rho R_H \\ F_5 &= \Lambda_v - \mu_v S_v - \Phi_2 S_v \\ F_6 &= \Phi_2 S_v - \mu_v I_v - \eta_2 I_v \\ F_7 &= \eta_2 I_v - \mu_v R_v \end{aligned} \right\} \quad (33)$$

The Jacobian computation is thus

$$J(S_H, E_H, I_H, R_H, S_v, I_v, R_v) \quad (34)$$

$$J = \begin{bmatrix} \frac{\partial F_1}{\partial S_H} & \frac{\partial F_1}{\partial E_H} & \frac{\partial F_1}{\partial I_H} & \frac{\partial F_1}{\partial R_H} & \frac{\partial F_1}{\partial S_V} & \frac{\partial F_1}{\partial I_V} & \frac{\partial F_1}{\partial R_V} \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\ \frac{\partial F_7}{\partial S_H} & \frac{\partial F_7}{\partial E_H} & \frac{\partial F_7}{\partial I_H} & \frac{\partial F_7}{\partial R_H} & \frac{\partial F_7}{\partial S_V} & \frac{\partial F_7}{\partial I_V} & \frac{\partial F_7}{\partial R_V} \end{bmatrix} \quad (35)$$

$$J = \begin{bmatrix} -(\mu_H + \Phi_1) & 0 & \frac{\beta_1 S_H}{(1+aI_H)^2} & \rho & 0 & \frac{\beta_2 S_H}{(1+aI_V)^2} & 0 \\ \Phi_1 & -(\mu_H + \tau) & \frac{\beta_1 S_H}{(1+aI_H)^2} & 0 & 0 & \frac{\beta_2 S_H}{(1+aI_V)^2} & 0 \\ 0 & \tau & -(\mu_H + \delta + \eta_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\mu_H + \rho) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\mu_V + \Phi_2) & \frac{\beta_3 S_V}{(1+aI_V)^2} & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 S_V}{(1+aI_V)^2} & -(\mu_V + \eta_2) & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta_2 & -\mu_V \end{bmatrix} \quad (36)$$

Now, at a disease-free equilibrium and introducing our 7 by 7 identity matrix, we have

$$|J - \lambda I| = \begin{vmatrix} -\mu_H - \lambda & 0 & \frac{\beta_1 \Lambda_H}{\mu_H} & \rho & 0 & \frac{\beta_2 \Lambda_H}{\mu_H} & 0 \\ 0 & -(\mu_H + \tau) - \lambda & \frac{\beta_1 \Lambda_H}{\mu_H} & 0 & 0 & \frac{\beta_2 \Lambda_H}{\mu_H} & 0 \\ 0 & \tau & -(\mu_H + \delta + \eta_1) - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\mu_H + \rho) - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_V - \lambda & \frac{\beta_3 \Lambda_V}{\mu_V} & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 \Lambda_V}{\mu_V} & -(\mu_V + \eta_2) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta_2 & -\mu_V - \lambda \end{vmatrix} \quad (37)$$

After solving and necessary simplifications of the 7 by 7 Jacobian matrix, we obtain the following eigenvalues:

$$\lambda_1 = -\mu_H, \lambda_2 = -\mu_V, \lambda_3 = -(\mu_H + \rho), \lambda_4 = -(\mu_H + \tau), \lambda_5 = -((\mu_H + \delta + \eta_1) - \frac{\tau \beta_1 \Lambda_H}{(\mu_H + \tau) \mu_H})$$

$$\lambda_6 = -(\mu_V + \eta_2) \text{ and } \lambda_7 = -\frac{\beta_3 \Lambda_V}{\mu_V (\mu_V + \eta_2)}$$

Note that $\lambda_5 = -((\mu_H + \delta + \eta_1) - \frac{\tau\beta_1\Lambda_H}{(\mu_H + \tau)\mu_H})$ can be re-written as

$$\lambda_5 = -(\mu_H + \delta + \eta_1)(1 - \frac{\tau\beta_1\Lambda_H}{\mu_H(\mu_H + \delta + \eta_1)(\mu_H + \tau)})$$

$$\lambda_5 \leq -(\mu_H + \delta + \eta_1)(1 - R_{OH})$$

Similarly, from $\lambda_7 = -\frac{\beta_3\Lambda_V}{\mu_V(\mu_V + \eta_2)} < 1$, the stability holds from λ_5 and λ_7 , which shows that $R_{OH} < 1$ and $R_{OV} < 1$. Therefore, $R_{HV} < 1$, which substantiates Theorem 1; therefore, the local asymptotical stability holds.

Every eigenvalue of the 7 by 7 dimensional systems is negative ($\lambda_1, \lambda_2, \lambda_3, \lambda_4, \dots, \lambda_7 < 0$), indicating that the local stability of the system DFE exists. $\square$

3.7. Global stability (GAS) of the Anthrax model at DFE

Theorem 3. If $R_{HV} < 1$, then model (11) is globally asymptotically stable (GAS) and unstable otherwise.

$$\left. \begin{aligned} D^\alpha S_H(t) &= \Lambda_H - \mu_H S_H - \Phi_1 S_H + \rho R_H \\ D^\alpha E_H(t) &= \Phi_1 S_H - \mu_H E_H - \tau E_H \\ D^\alpha I_H(t) &= \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\ D^\alpha R_H(t) &= \eta_1 I_H - \mu_H R_H - \rho R_H \\ D^\alpha S_V(t) &= \Lambda_V - \mu_V S_V - \Phi_2 S_V \\ D^\alpha I_V(t) &= \Phi_2 S_V - \mu_V I_V - \eta_2 I_V \\ D^\alpha R_V(t) &= \eta_2 I_V - \mu_V R_V \end{aligned} \right\} \quad (38)$$

We start by implementing the method in [35, 45], dividing the system into two distinct parts and also declaring some new variables to verify Theorem 2.

In addition, $E^0 = (S_H^0, E_H^0, I_H^0, R_H^0, S_V^0, I_V^0, R_V^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0 \right)$ is a necessary condition for anthrax-free equilibrium.

Dividing the system, we have

$$F(K, J) = \begin{bmatrix} \Lambda_H - \mu_H S_H - \Phi_1 S_H + \rho R_H \\ \eta_1 I_H - \mu_H R_H - \rho R_H \\ \Lambda_V - \mu_V S_V - \Phi_2 S_V \\ \eta_2 I_V - \mu_V R_V \end{bmatrix}, \quad G(K, J) = \begin{bmatrix} \Phi_1 S_H - \mu_H E_H - \tau E_H \\ \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\ \eta_2 I_V - \mu_V R_V \end{bmatrix} \quad (39)$$

where $K = (S_H, R_H, S_V, R_V) \in R_+^4$ and $J = (E_H, I_H, I_V) \in R_+^3$, K denoting the set of uninfected and J denoting the set of infected. We say that the point $E^0 = (K^*, 0)$ is globally asymptotically stable (GAS) if $R_{HG} < 1$, $K^* = \left(\frac{\Lambda_H}{\mu_H}, 0, \frac{\Lambda_V}{\mu_V}, 0 \right)$, denoting the disease-free equilibrium of the subpopulation.

The subsequent conditions hold the following:

$\frac{d^\alpha K}{dt^\alpha} = F(K, 0)$, then E^0 is GAS; here, $0 < \alpha \leq 1$, which is the integer order of our fractional model, and we assume for simplicity that $\alpha = 1$

There exist $G(K, J) = BC - G^0(K, J)$ such that $G^0(K, J) \geq 0$, for every $(K, J) \in R_+^7$

Proof. Proof of the first conditions is as follows:

$\frac{dK}{dt} = (K, 0)$, for $J = 0$, so we have that the infection does not exist in this instance. Remember, J denotes the infected classes, and the system is reduced to

$$F(K, 0) = \begin{Bmatrix} \Lambda_H - \mu_H S_H + \rho R_H \\ -\mu_H R_H - \rho R_H \\ \Lambda_V - \mu_V S_V \\ -\mu_V R_V \end{Bmatrix} \quad (40)$$

It is evident that the first condition holds because this has been obtained earlier while proving the positivity of the model (using Mittag-Leffler); however, the below proof also suffices.

Mini-proof.

$$\frac{dS}{dt} = (\Lambda_H + \rho R_H) - \mu_H S_H, \text{ using the method of } I.F = e^{\int \mu_H dt}$$

$$\frac{d(S_H e^{\mu_H t})}{dt} = (\Lambda_H + \rho R_H) e^{\mu_H t}$$

$S_H = \frac{\Lambda_H}{\mu_H} + \frac{1}{e^{\mu_H t}} \int \rho R_H e^{\mu_H t} dt$, and as $t \rightarrow \infty$, we have that $S_H(t) \rightarrow \frac{\Lambda_H}{\mu_H}$. This also goes for the vector population:

$S_V(t) \rightarrow \frac{\Lambda_V}{\mu_V}$ as $t \rightarrow \infty$, showing that both populations are bounded.

The reduced system for the GAS equilibrium point is $K^* = \left(\frac{\Lambda_H}{\mu_H}, 0, \frac{\Lambda_V}{\mu_V}, 0 \right) = (S_H, R_H, S_V, R_H,)$

Conveying the global convergence and global stability on our model, $F(K, 0) \in R_+^7$

This completes the mini-proof. $\square$

Proof. Proof of the second condition.

We need to show that for every $G^0(K, J) \geq 0$, $(K, J) \in R_+^7$, $G(K, J) = BC - G^0(K, J)$.

We have

$$G(K, J) = \frac{dY}{dt} \left\{ \begin{array}{l} \Phi_1 S_H - \mu_H E_H - \tau E_H \\ \tau E_H - \mu_H I_H - \delta I_H - \eta_1 I_H \\ \Phi_2 S_V - \mu_V I_V - \eta_2 I_V \end{array} \right\} \quad (41)$$

which yields

$$B = \begin{bmatrix} -(\mu_H + \tau) & 0 & 0 \\ \tau & -(\mu_H + \delta + \eta_1) & 0 \\ 0 & 0 & -(\mu_V + \eta_2) \end{bmatrix} \quad (42)$$

Clearly, all diagonal entries of B are less than unity (i.e., 1) after differentiating with respect to variables in matrix C.

$C = \begin{bmatrix} E_H \\ I_H \\ I_V \end{bmatrix}$, where $G(K, J) = G^0(K, J)$ is the stable state matrix, which establishes the second

condition of the GAS.

$$BC = \begin{bmatrix} -(\mu_H + \tau) & 0 & 0 \\ \tau & -(\mu_H + \delta + \eta_1) & 0 \\ 0 & 0 & -(\mu_V + \eta_2) \end{bmatrix} \begin{bmatrix} E_H \\ I_H \\ I_V \end{bmatrix} \quad (43)$$

$$G^0(K, J) = \begin{bmatrix} \Phi_1\left(\frac{\Lambda_H}{\mu_H} - S_H\right) \\ 0 \\ \Phi_2\left(\frac{\Lambda_V}{\mu_V} - S_V\right) \end{bmatrix} \quad (44)$$

which demonstrate the boundedness of our model, which also substantiate Theorem 2.

This completes the proof. $\square$

3.8. Analysis of the sensitivity of parameters of the Anthrax–Vector model

The main goal of the sensitivity analysis is to ascertain how each variable affects the rate of reproduction. This aids in determining the parameters that have a significant influence on R_{HV} . In order to determine whether or not immunization, mortality, and infection treatments could help in the management or eradication of the disease in the system, the basic reproduction rate is often examined [46]. This technique is widely employed to test the resilience of model assumptions to variable values. The examination of the model's reproduction number R_{HV} , which looks for variance and the impact of changing a parameter on R_{HV} , will be carried out.

Definition 7. *The normalized forward sensitivity index of a variable a is given by*

$$M_b^a = \frac{\partial a}{\partial b} \times \frac{b}{a} \quad (45)$$

which depends on the differentiability of parameter b .

We would now have to carry out the analysis of the fundamental reproduction number specifically on some key parameters that make up the reproduction number R_{0HV} and with regard to parameters that make up our model.

The sensitivity index for $\beta_1, \beta_2, \beta_3, \dots$ and every other parameter will be computed for as follows:

$$R_{0HV} = \sqrt{\frac{\beta_1 \beta_3 \Lambda_V \Lambda_H \tau}{\mu_V \mu_H (\mu_V + \eta_2) (\mu_H + \delta + \eta_1) (\mu_H + \tau)}} \quad (46)$$

Analyzing the derivatives in Equation (47) gives

$$M_{\beta_1}^{R_{HV}} = \frac{\partial R_{HV}}{\partial \beta_1} \cdot \frac{\beta_1}{R_{HV}}, \quad \frac{\partial R_{HV}}{\partial \beta_1} = 0.5 \frac{R_{HV}}{\beta_1}, \text{ hence}$$

$$M_{\beta_1}^{R_{HV}} = \frac{\partial R_{HV}}{\partial \beta_1} \cdot \frac{\beta_1}{R_{HV}} = 0.5 \frac{R_{HV}}{\beta_1} \cdot \frac{\beta_1}{R_{HV}}$$

Conclusively,

$$M_{\beta_1}^{R_{HV}} = +0.5$$

With this, we obtain the sensitivity index for β_1 .

In the same vein, we will use this method for all other parameters that make up the reproduction number to obtain their sensitivity index, though not all the parameters have positive values according to Table 2.

Table 2. Parameters and their sensitivity indices (SI).

Parameters	Sensitivity Indices (S.I)
Λ_H	+ve
Λ_V	+ve
μ_H	-ve
μ_V	-ve
β_1	+ve
β_3	+ve
β_2	+ve
η	-ve
δ	-ve
τ	+ve

3.9 Interpretation of R_{HV} sensitivity indices

The value of R_{HV} in epidemiological models indicates how easily a disease may spread throughout the community. By calculating the sensitivity indices of R_{HV} with regard to the model's parameter values, we will be able to calculate the decrease in infections caused by the diseases. The contribution of each parameter to the dynamics of the diseases is determined by the sensitivity indices. We determine the sensitivity of R_{HV} to each parameter in model (11), taking into account all of the system's properties.

Table 2 lists the sensitivity indices of R_{HV} for each system parameter in model (11).

A thorough sensitivity analysis of R_{HV} revealed that a rise in η would result in a reduction in R_{HV} . Furthermore, if η were to decrease, R_{0HV} would rise. Moreover, a rise in R_{0HV} implies that there is an increase in the values of $\Lambda_H, \Lambda_V, \beta_1, \beta_2, \text{ and } \beta_3$, and a fall in R_{0HV} would result from a decrease in those values, respectively.

4 Numerical simulations

We have reviewed, investigated, and evaluated the analytical/simulated outcomes of the anthrax–vector interactive model in this section. This is completed to confirm the results of our sensitivity analysis, to validate our theoretical conclusions, and to investigate, evaluate, and elucidate the dynamic behavior of the infection in the proposed population. We have simulated the full model and individual compartments using the MATLAB program, taking into account the initial circumstances (state variable values) and changing a few significant model parameters. Tables 3 and 4 present the values and origins of these parameters. Furthermore, the extended Adams–Bashforth–Moulton-type predictor–corrector technique is used to obtain approximate solutions for our fractional ordered model using MATLAB [47]. Time series solutions of system (11) with varying values of a are produced using the same parameters given in Table 4 for the population (6000, 1750, 700, 150) for the human population and (100, 70, 0) for the vector population (refer to Figures 14–20) for the simulated results.

Table 3. State variables values and their sources used in simulations.

State Variable	Values	Source
S_H	6000	Assumed
E_H	1750	Assumed
I_H	700	Assumed
R_H	150	Assumed
S_V	100	Assumed
I_V	70	Assumed
R_V	0	Assumed

Table 4. Parameter values and their sources used in simulation.

Parameter	Values	Source
Λ_H	0.2	[25]
Λ_V	0.005	[25]
μ_H	0.0003466	Assumed
μ_v	0.00261076	Assumed
δ	0.01710615	Assumed
β_1	0.04	Assumed
β_2	0.04	Assumed
β_3	0.005	Assumed
Φ_1	0.091193	Assumed
Φ_2	0.1	Assumed

ρ	0.0090	Assumed
η_1	0.30	Assumed
η_2	0.1	[26]
τ	0.057	[26]
a_H	0.95	Assumed
a_V	0.95	Assumed

While some of these parameters were based on reasonable estimations and assumptions, the majority of them were clinical facts that were taken from reputable studies.

The above tables were derived from Table 1.

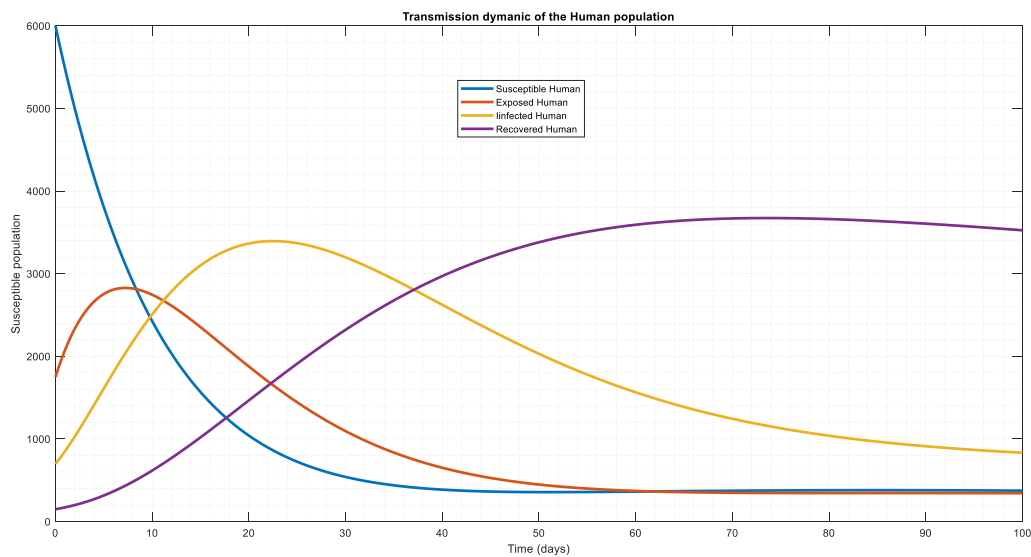


Figure 2. Solution trajectories of the susceptible human population.

The human population dynamical behavior with $\Lambda_H = 0.2$, $\Phi_1 = 0.091193$, $\mu_H = 0.0003466$, $\rho = 0.0085$, $\tau = 0.10$, $\delta = 0.01710615$, and $\eta_1 = 0.30$ demonstrate the coexistence of the compartment and drive the system's tendency toward an endemic condition.

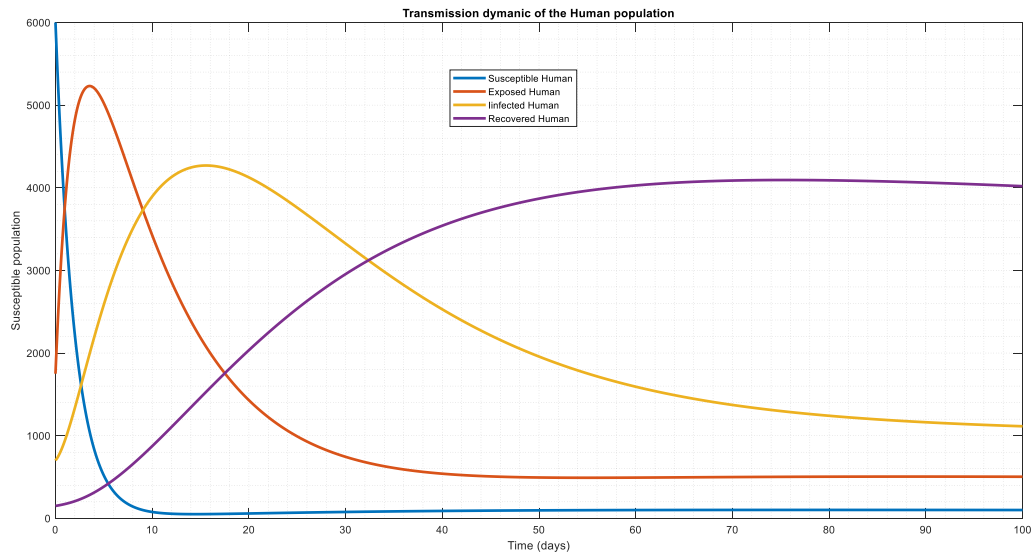


Figure 3. Solution trajectories of the susceptible human population with variations of a few parameters.

Here, the recruitment was increased $\Lambda_H = 12$ and the force of infection $\Phi_1 = 0.5$, while other parameters remain the same, which also shows that all compartments co-exist, causing the population to approach an endemic state, but one thing that is noticeable here is that the susceptible population shows a sharp decline compared to Figure 2.

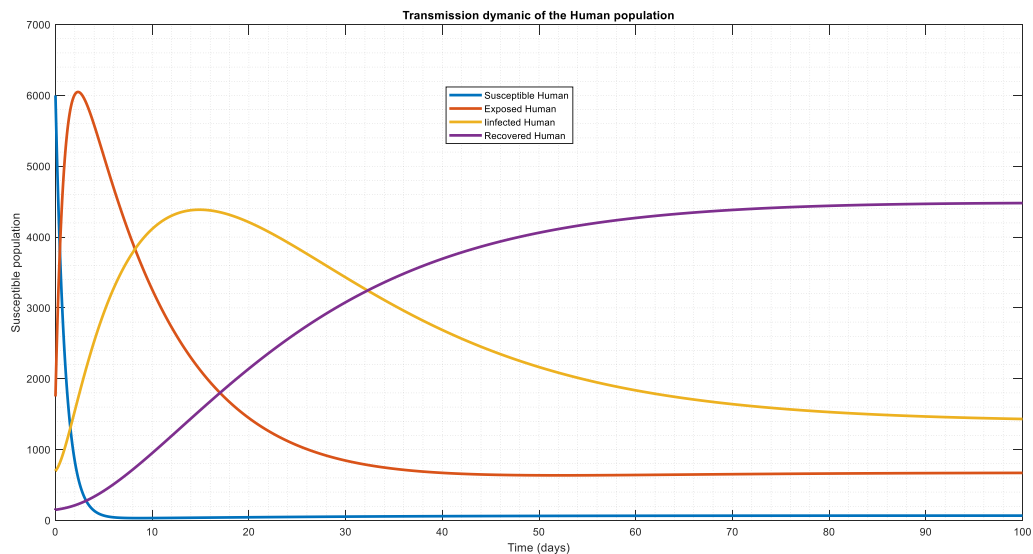


Figure 4. Solution trajectories of the susceptible human population with variations of a few parameters.

Similarly, the recruitment rate was increased a third time, i.e., $\Lambda_H = 25, \Phi_1 = 1$, and again, we notice a sharp decline in the susceptible region, which will hence affect the exposed and the infected, causing the whole population to tend to toward an endemic state.

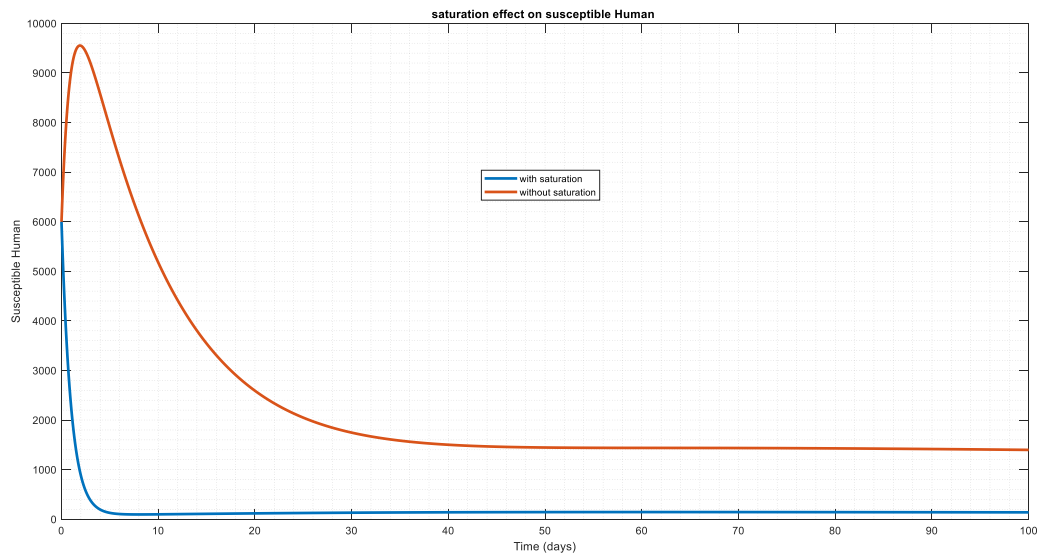


Figure 5. Solution trajectories of the susceptible human population with(out) the saturation factor.

The susceptible humans' changing effects, both with and without the “saturation factor”, indicate that the introduction of saturation lowers the incidence of human vulnerability to anthrax.

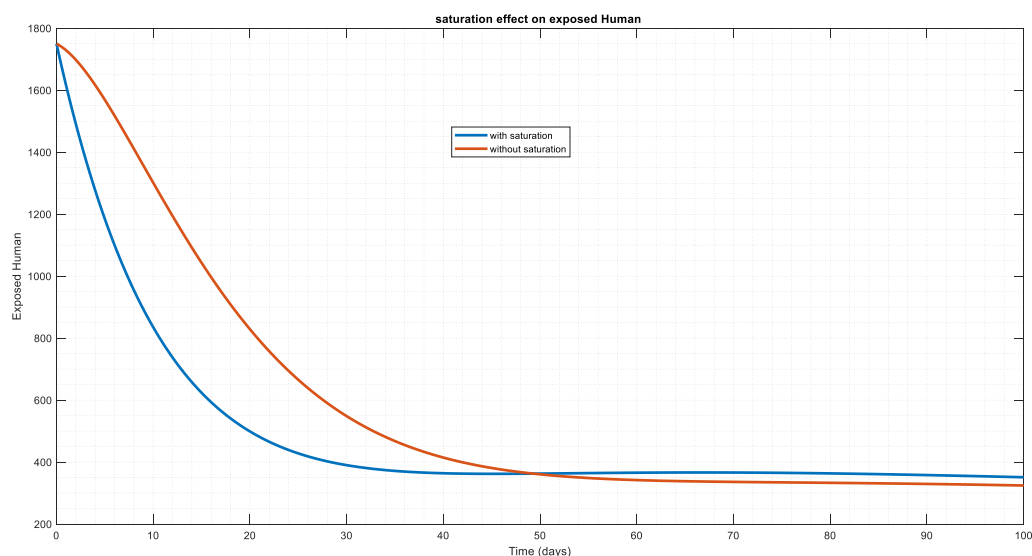


Figure 6. Solution trajectories of the exposed human population with(out) the saturation factor.

The exposed humans' changing effects, both with and without the “saturation factor”, indicate that the introduction of saturation lowers the incidence of exposed compartments to anthrax.

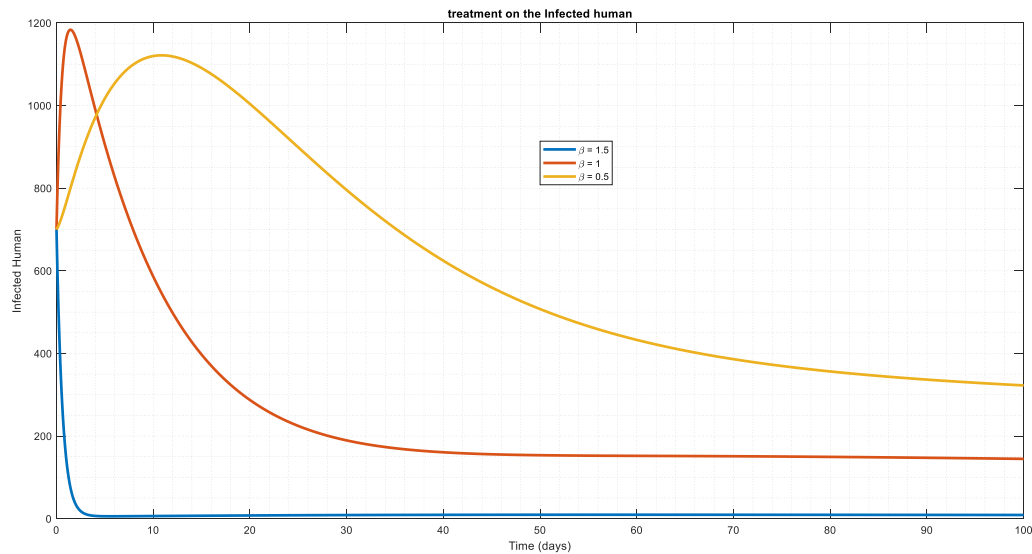


Figure 7. Solution trajectories of the infected human population with proper treatment by doubling and tripling the effect.

The effect of increasing the rate of treatment for infected individuals using vaccination administration (along with other essential treatments) demonstrates how early antiviral medication administration and implementation may significantly reduce the population of the infected in the infected human compartment.

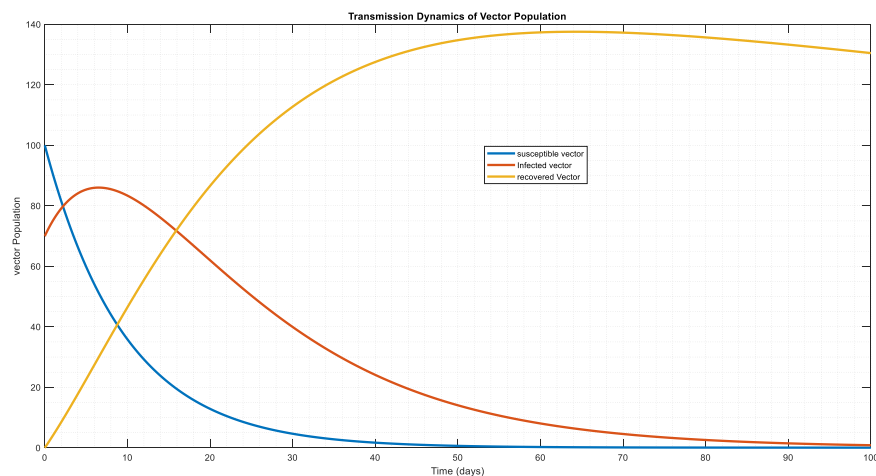


Figure 8. Solution trajectories of the susceptible vector.

The dynamical behavior of the vector population with $\Lambda_V = 0.005$, $\Phi_2 = 0.1$, $\delta = 0.00261076$, $\eta_2 = 0.057$ shows how the compartments co-exist, in which, in essence, the vector population tends towards an endemic state.

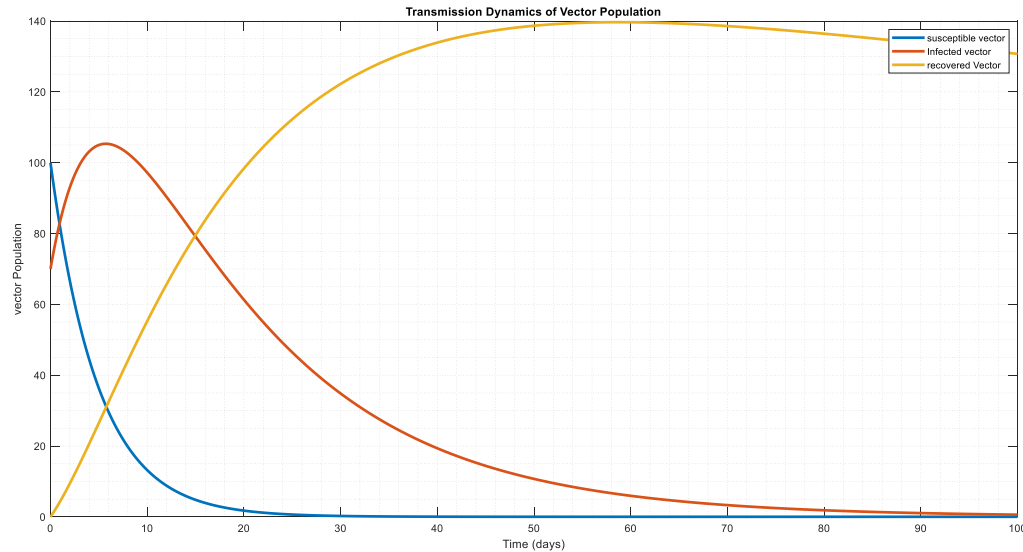


Figure 9. Solution trajectories of the susceptible vector while doubling the force of infection.

In the above figure, $\Phi_2 = 0.2$, we doubled the force of infection; while other parameters remain the same, we notice a sharp decline in the susceptible class, which changes the behavior of the population as a whole.

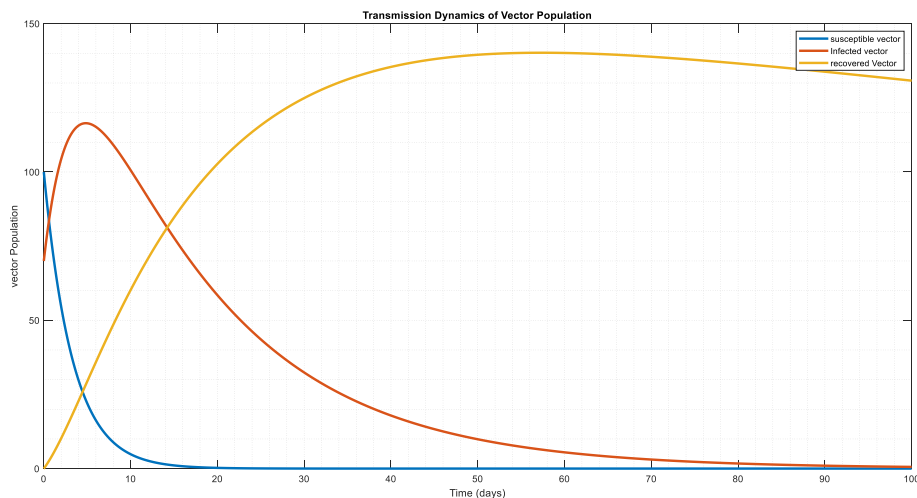


Figure 10. Solution trajectories of the susceptible vector while tripling the force of infection.

In the above figure, $\Phi_2 = 0.3$ to see how the population behaves; while other parameters remain same, we notice a sharp decline in the susceptible class, which changes the behavior of the population as a whole.

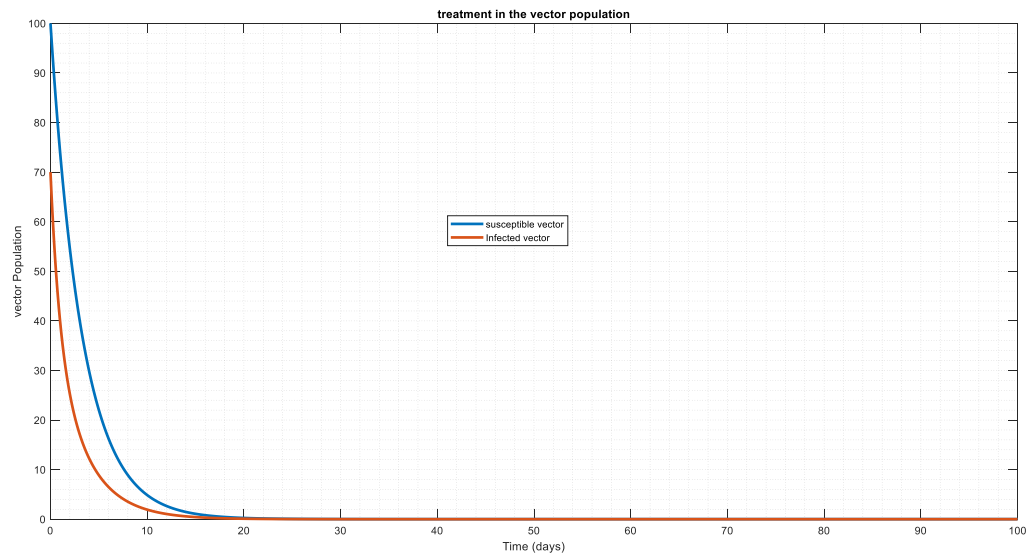


Figure 11. Solution trajectories of the susceptible and the infected vector while implementing the control plan.

Here, we try to introduce an effective control plan to the vector population (domestic), with $\Lambda_v = 0.005$, $\Phi_2 = 0.3$. The figure above shows that the vector population can approach a disease-free equilibrium (DFE), especially the susceptible and infected populations.

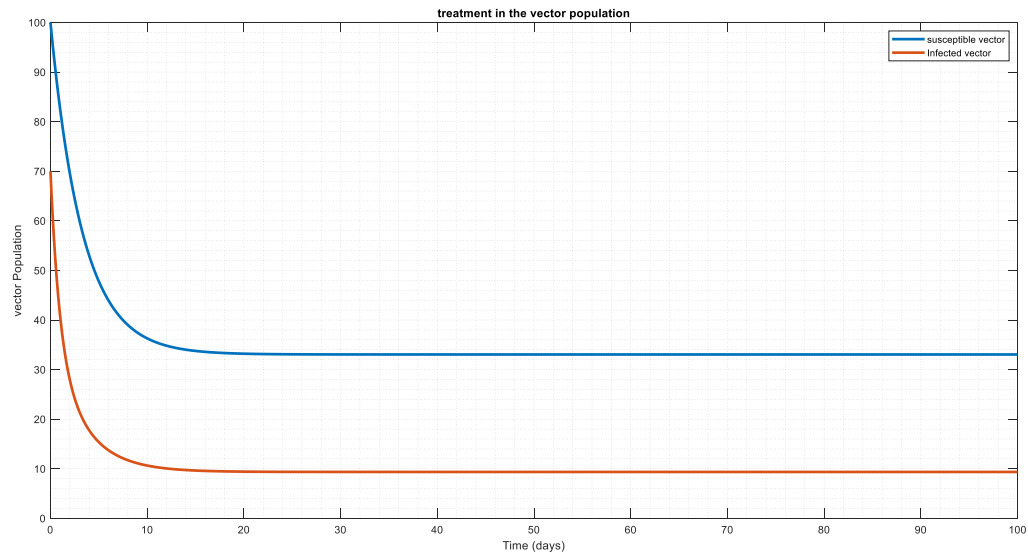


Figure 12. Solution trajectories of the susceptible and the infected vector while implementing a control plan by increasing the recruitment rate.

In the figure above, the vector recruitment rate was increased to $\Lambda_v = 10$; while other parameters remain the same, we noticed that, irrespective of the susceptible number to the system, with effective control strategies, the vector population will still attain a DFE.

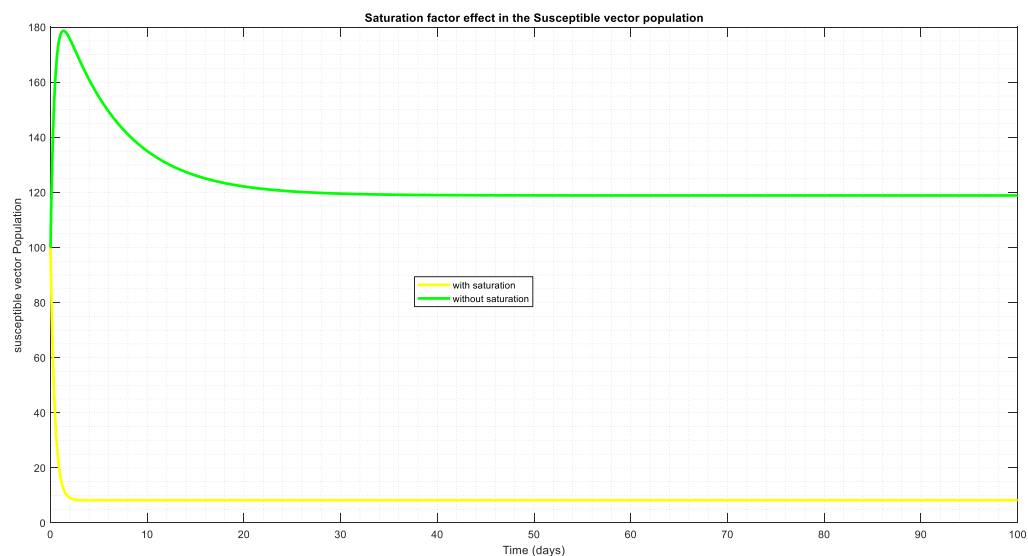


Figure 13. Solution trajectories of susceptible vector population with(out) saturation.

The susceptible vector changing effects, both with and without the “saturation factor”, indicate that the introduction of saturation lowers the incidence of vector vulnerability to anthrax.

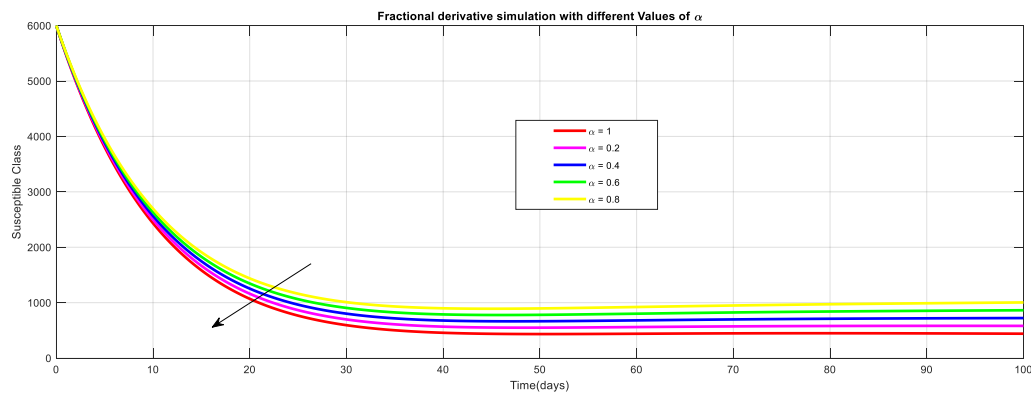


Figure 14. The fractional derivative variation simulation on susceptible human populations.

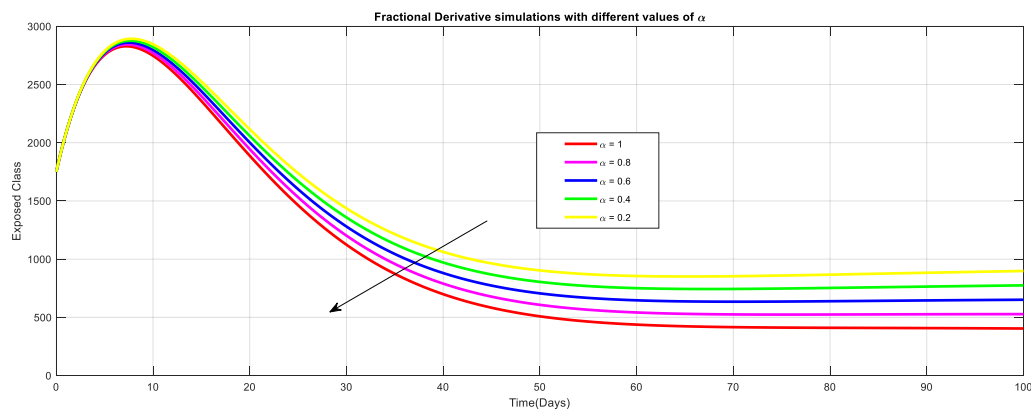


Figure 15. The fractional derivative variation simulation on exposed human populations.

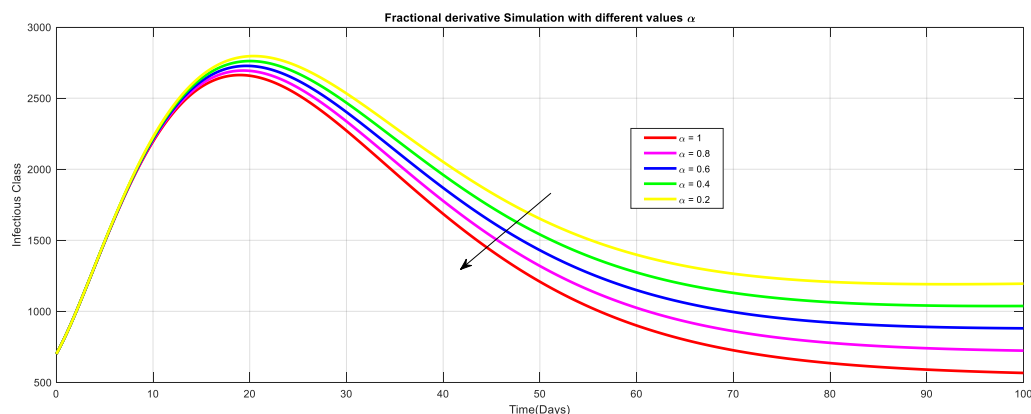


Figure 16. The fractional derivative variation simulation on infected human populations.

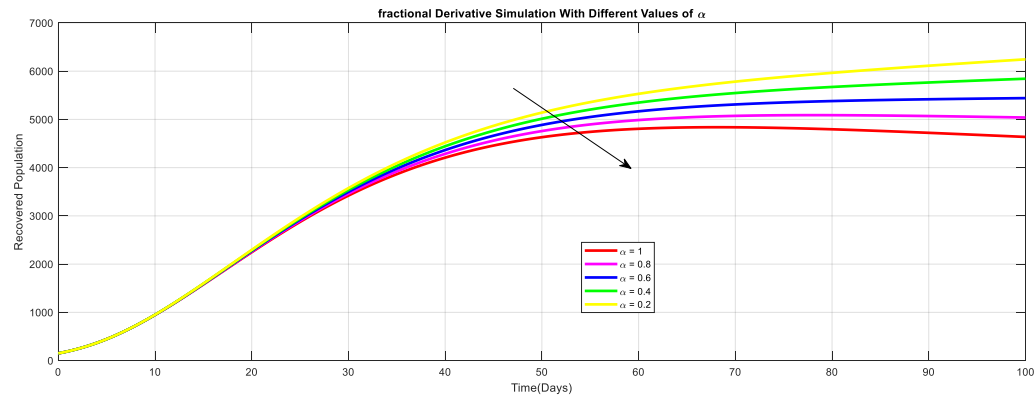


Figure 17. The fractional derivative variation simulation on recovered human populations.

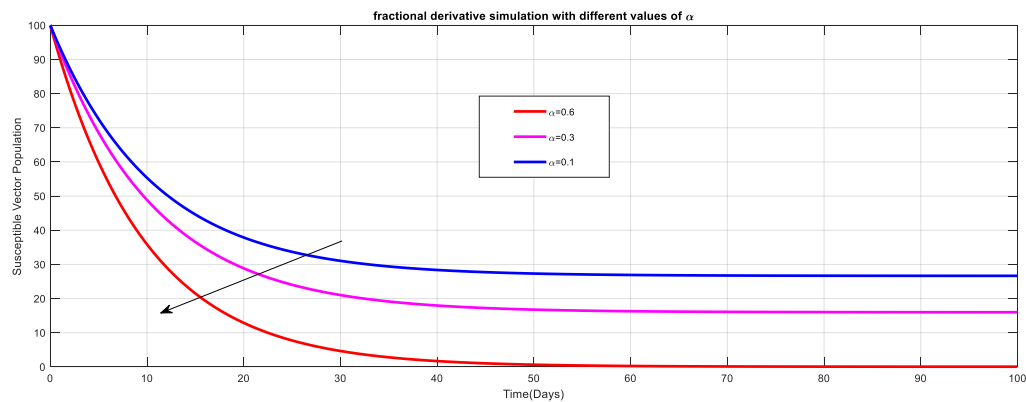


Figure 18. The fractional derivative variation simulation on susceptible vector populations.

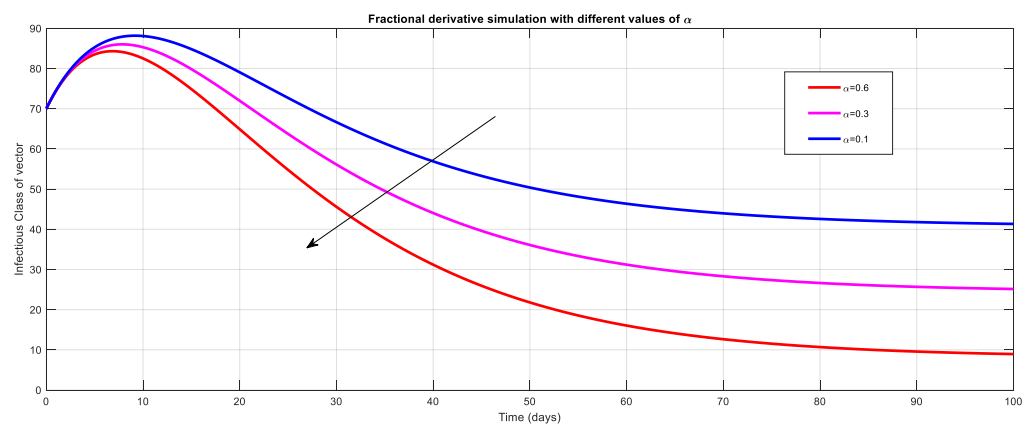


Figure 19. The fractional derivative variation simulation on infected vector populations.

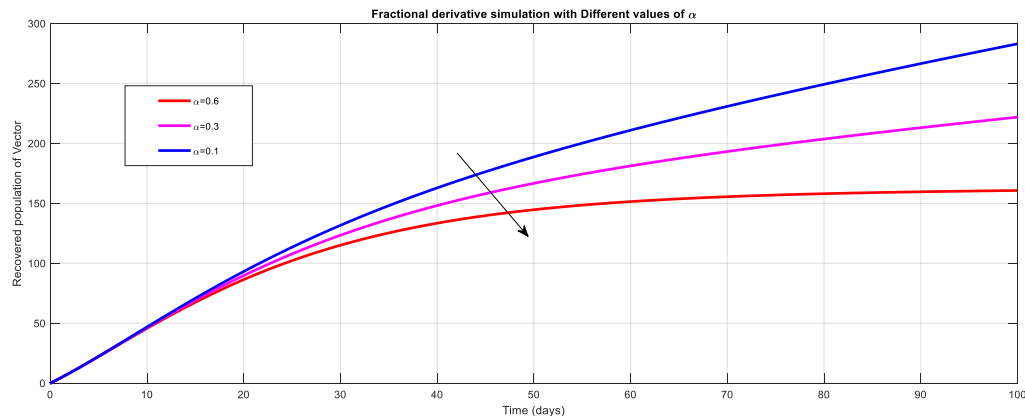


Figure 20. The fractional derivative variation simulation on recovered vector populations.

5. Discussions and conclusion

Using the values on Tables 3 and 4 alongside the parameters, we have simulated the model. We now must adjust a few of these factors on the above table in order to see the changes (behavior) in our model.

Using the initial conditions and parameter values from Tables 3 and 4, the merged graphs of the whole human population are shown in Figure 1. The section of exposed and infected humans increases during the starting phase (instantaneous time); however, over time, both the exposed and the infected reach a stable state as a result of the system's dynamic behavior, as shown in Figure 1. The human population begins to recover after a while; however, during the early phase, the whole system tends to be vulnerable, demonstrating the mathematical epidemiology of our model (which tends towards endemic conditions).

The dynamic behavior of each human compartment is depicted in Figure 3 when the infection force Φ_1 and the human immigration rate Λ_H are varied. By changing these parameters, we found that people become increasingly exposed to the virus and become sick; the population of susceptible people declines dramatically and abruptly in less than ten (10) days compared to Figure 1, although people generally recover within ninety days. These all demonstrate how the system is approaching an endemic condition since every compartment coexists in it.

The system's behavior when the immigration rate is raised and the infection force is $\Phi_1 = 1$ is unity, as seen in Figure 4. The human system is exposed very quickly at one point in time, again demonstrating the mathematical epidemiology of our model, and after a month (30 days), the infected human population likewise grows at a steady rate, leading to a sharp decline in the susceptible population below 10 days.

In Figures 5 and 6, the dynamic of the exposed and susceptible humans reveals that when "saturation" occurs, the populations of these humans are drastically reduced, leading the system to approach stability (DFE). This could be caused by various control measures, such as hunting wild animals with the infection in the instance of the vector population, as seen in Figure 13;

immune responses to the infection; or the early management of medications, such as vaccination (and other necessary interventions) in both populations.

For the vector system's dynamic behavior in Figures 8–10, in relation to the starting conditions and parameter figures on Tables 3 and 4, we double and triple the infection force for Figures 9 and 10, respectively. This indicates that the vector tends to be exposed at a single moment, resulting in infection in lesser periods of 30, 20, and 10 days apart; however, the population of the infected eventually reaches a stable state, and the population of the susceptible disappear over time, leaving only the population of the infected in the system.

Figure 7 presents a control strategy that aims to flatten the spread of disease among the infected human population. The therapy is modified by doubling and tripling its effect over time; hence, the system tends towards a DFE state. That is a disease-free system.

Since vectors are the carriers of the disease, we aim to minimize the rate at which humans will contract it by introducing an effective control plan in susceptible vector and infected vector populations (Figures 11 and 12). Additionally, Figure 12 illustrates that, regardless of the number of susceptible vector populations, an effective control plan will eventually cause the population to reach a disease-free system.

Figures 14–20 illustrates the constantly changing complexity of the fractional ordinary differential equations, highlighting their enhanced capability as descriptors for biological phenomena in comparison to the ordinary differential equation models. Additionally, the model's solutions, which depend on the continuously changing time-fractional derivative, consistently converge to equilibrium points, which in turn aid the stability in each simulated compartment.

5.1 Conclusion

The present study examines an unprecedented interactive anthrax–vector model, constructed on a seven-dimensional nonlinear system of fractional differential equations, with the aim of characterizing and capturing the dynamics of the anthrax infection's propagation. Stability, boundedness, positivity, and sensitivity analyses have been provided for this model. Importantly, we have used clinical, approximated, and assumed data from the well-known literature to run the model.

We draw the conclusion from our data and research that the system approaches an anthrax-free equilibrium instantaneously when “saturation” occurs (which may be caused by treatments or control measures impeding the virus's spread in a vulnerable population). In other words, the mass elimination of the disease would be greatly aided by the execution of a suitable management strategy (treatment and other medical interventions).

Furthermore, based on the stability and sensitivity study, the vector recruitment rate Λ_V and the effective contact rates ($\beta_1, \beta_2, \beta_3$) that result in anthrax infection are the most sensitive parameters that need to be addressed by the control organization (health officials). Because of this, the regulated basic reproduction number would continue to be less than unity (1).

Additionally, we recommend that early intervention and prompt treatment with a “recommended diagnosis” be used to increase the effectiveness and expand the capacity of

treatment in order to instantly eradicate the disease. People should also make every effort to maintain maximum hygiene and a clean environment in order to avoid providing a breeding ground for *Bacillus anthracis* within homes and among animal populations (domestic); this could be accomplished through public awareness campaigns and sensitization to the anthrax infection.

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