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Modeling of Anthrax Disease via Efficient Computing Techniques

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Abstract: Computer methods have a significant role in the scientific literature. Nowadays, development in computational methods for solving highly complex and nonlinear systems is a hot issue in different disciplines like engineering, physics, biology, and many more. Anthrax is primarily a zoonotic disease in herbivores caused by a bacterium called *Bacillus anthracis*. Humans generally acquire the disease directly or indirectly from infected animals, or through occupational exposure to infected or contaminated animal products. The outbreak of human anthrax is reported in the Eastern Mediterranean regions like Pakistan, Iran, Iraq, Afghanistan, Morocco, and Sudan. Almost ninety-five percent chances are the transmission of the bacteria from forming spores by the World Health Organization (WHO). The modeling of an anthrax disease is based on the four compartments along with two humans (susceptible and infected) and others are dead bodies and sporing agents. The mathematical analysis is studied along with the fundamental properties of deterministic modeling. The stability of the model along with equilibria is studied rigorously. The authentication of analytical results is examined through well-known computer methods like Euler, Runge Kutta, and Non-standard finite difference (NSFD) along with the feasible properties (positivity, boundedness, and dynamical consistency) of the model. In the end, comparison analysis of algorithms shows the effectiveness of the methods.

Keywords: Anthrax disease; deterministic modeling; stability analysis; computer methods

1 Introduction

The major cause of many diseases in the world is viruses and bacteria. Anthrax is a bacterial disease. Anthrax is a disease of herbivores such as sheep, goats, horses, cows and transmitted to humans. Infected Animals are the source to transmit the bacteria in humans. It does not spread from animal to animal or man to man like covid 19. Anthrax spores can enter the human body through via breathing and cuts on the skin. Its incubation period varies

from one to several days. Insects are also the source of the transmission of anthrax. Bacteria are prokaryotes that contain a cell wall, capsule, ribosomes, pili, flagella, and DNA. This species of a bacterium can survive in a harsh environment. Bacteria are considered the eldest living organisms on the earth. The Greek word anthrax means coal. Egypt is the region of anthrax disease. Pieces of evidence show that many scholars of Greece and Rome are well known about anthrax. Anthrax disease is the cause of the downfall of Rome. Those animals which become the victims of anthrax do not show any proper symptoms. They die after infection without leaving diagnostics symptoms. Three forms of anthrax are cutaneous, inhalation, and gastrointestinal. Cutaneous anthrax is about 95% in society. It is a very less dangerous form of anthrax. This form of anthrax usually shows its symptoms for 1 to 7 days. A person can get cutaneous anthrax through cuts on the skin. The persons who are working in tanning and woolen industries can easily get cutaneous anthrax. This form of anthrax is most common in the hands, foot, and neck regions. The common symptoms of cutaneous anthrax are small blisters appear on the skin, looks like an insect bite, swelling in tissues around the sore, a black-centered painless sore appears on the skin such as on the face, hands, and neck regions. We can easily survive by using proper antibiotics. Inhalation anthrax is the most dangerous form of anthrax. It usually shows its symptoms within a week and can become chronic. Its symptoms are fever, feeling cold disorder in the chest, shortness of breath, coughing, vomiting, headache, pain in the stomach, and fatigue. No evidence in the world shows its propagation with infected milk. There is a disorder in the elementary canal on ingesting. sixty percent of patients can easily survive on the proper treatment of gastrointestinal anthrax. The death ratio of humans via anthrax is 20% (cutaneous), 75% (gastrointestinal), and 80% (lung infection). This disease is common in developing countries like the Sahara Desert in Africa, central and southwestern Asia (Turkey, Labnan, Syria, Iran, Egypt), and the Caribbean (Belarus Hungry, Romania, Slovakia). Dozens of cows are died due to anthrax in Pakistan. Mackey et al. studied a model in which describes the transmission of disease in zebras [1]. Baloba et al. investigated the control measures or mechanisms to overcome anthrax [2]. Stella et al. suggested the SIR model for anthrax disease in the animal community [3]. Rezapour et al. proposed a mathematical model for explaining the spread of anthrax with the help of the Caputo-Fabrizio technique [4]. Croicu suggested the IACN model of anthrax disease in herbivores [5]. Gomez et al. suggested the SIML model for transmission of anthrax [6]. Mushayabasa et al. proposed the SEIPC model with the well-known assumptions of mathematics to dispose of carcasses [7]. Roy et al. suggested the SIAC model for the spread of anthrax in animal populations [8]. Mushayabasa suggested the model and its reproduction number with the help of the Lyapunov method [9]. Wanying et al. suggested different factors for controlling the death rate during the bioterrorist attack of anthrax [10]. Pantha et al. studied optimal control of a mathematical model for the spread of anthrax [11]. Gutting studied the inhalation of anthrax in rabbits [12]. Toth et al. estimate the incubation period for the treatment of inhalation anthrax in humans [13]. Day et al. present a model for control of inhalation anthrax [14]. Wilkening proposed an incubation period and describe the importance of the incubation period for controlling inhalation anthrax [15]. Li et al. studied a dynamical mathematical model for the analysis of anthrax disease [16]. Pittman described the importance of the anthrax vaccine in humans [17]. Furniss et al. suggested a model for the control of anthrax in Kruger national park [18]. Pauline described the significance of modeling diseases [19]. Helikumi et al. described the importance of reproduction numbers in modeling various diseases like anthrax [20]. Webb proposed the risk of anthrax in biowarfare [21]. Afshar et al. discussed the detail of a clinical case of anthrax in Iran [22]. Hashemi et al. argued the gastrointestinal patient of anthrax in northeast Iran [23]. Osman et al. developed a model for Listerriosis and anthrax [24]. Brookmeyer et al. suggested the incubation period of anthrax, and they also discussed the incubation period depends on the age and amount of spore that enters in lungs [25]. Karginov et al. discussed that ciprofloxacin with antibodies and the growth of bacteria should be efficient for curing anthrax [26]. Loving et al. described that mice species were more efficient for the study of anthrax [27]. Radosavljevic et al. suggested the curing techniques for the bioterrorist attack of anthrax [28]. Mathematical techniques were studied to analyze the transmission of infectious diseases [29–42]. In this paper, we study the dynamics of anthrax disease via computational methods. We can observe that computational methods in literature have

many problems like negativity, unboundedness, and inconsistency of solutions. These issues will resolve by our proposed idea that is a non-standard finite difference method (NSFD). Also, NSFD fulfills the properties of the biological problem. The rest of the paper is styled as follows: In Section 2 the modeling of anthrax is defined. In Section 3 the construction way of the anthrax epidemic model, equilibrium points and computer methods and their convergence are explained. In the last section conclusion and future problems are discussed.

2 Modelling of Anthrax

By using the theory of population dynamics of epidemiological type, susceptible and infected is the sum of the total population. The evolution process of the bacteria describes the effect of the epidemiological parameters based on the system of nonlinear coupled ordinary differential equations. Thus, a continuous model for populations regarding anthrax is described in Fig. 1.
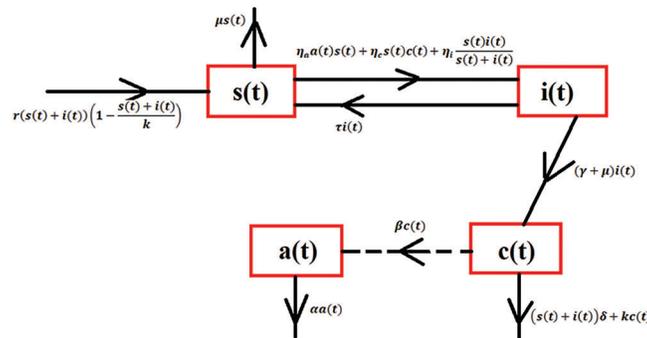
Figure 1: Transmission map of anthrax

The vertical transmission of humans, birth and death rates of humans are considered approximately equal. The physical relationship of variables and constants are shown in Tab. 1 as follows:

Table 1: Physical relevance of the model

Parameters	Descriptions
$s(t)$	Susceptible population.
$i(t)$	Infected population
$c(t)$	Infected dead bodies
$a(t)$	The number of spores in grams.
r	Birth rate/day.
μ	Death rate/day.
K	The capacity of infection.
η_a	Rate of spread of disease from the environment to susceptible.
η_i	Rate of spread of disease from infected to susceptible.
η_c	Rate of spread of disease from carcasses to susceptible.
τ	Recovery rate / day.
γ	Disease death rate / day.
δ	Carcass decomposition rate / day / animal.
k	Decomposition rate of carcasses.
β	Growth rate of spore rate / day / carcasses.

The system of differential equations can be deriving from the above flow chart of population as follows:

$$\frac{ds}{dt} = \Lambda - \eta_a as - \eta_c cs - \eta_i is - \mu s + \tau i, t \geq 0 \tag{1}$$

$$\frac{di}{dt} = \eta_a as + \eta_c cs + \eta_i is - (\gamma + \mu + \tau)i, t \geq 0 \tag{2}$$

$$\frac{da}{dt} = -\alpha a + \beta c, t \geq 0 \tag{3}$$

$$\frac{dc}{dt} = (\gamma + \mu)i - \delta c - kc, t \geq 0 \tag{4}$$

The system (1-4) is based on the feasible region of the model as follows:

$$Z = \left\{ (s, i, a, c) \varepsilon R_+^4 : s \geq 0, i \geq 0, a \geq 0, c \geq 0 : s + i \leq \frac{\Lambda}{\mu}, c \leq \frac{\mu(\gamma + \mu)}{\delta \Lambda + \mu k}, a \leq \frac{\mu(\gamma + \mu)}{\alpha \Lambda + \mu k} \right\}.$$

Lemma 1: The solutions $(s, i, a, c) \varepsilon R_+^4$ of the system (1-4) are positive at any time $t \geq 0$, if the rate of change of state variable is non-negative at the trivial stage.

Proof. In this section, we prove the positivity of the first two compartments due to human populations. Forgiven an initial condition $s \geq 0, i \geq 0$,

From Eq. (1),

$s' = \Lambda - \eta_a as - \eta_c cs - \eta_i is - \mu s + \tau i, s' \geq (-\eta_a a - \eta_c c - \eta_i i - \mu)s.$

$lns \geq -(\eta_a a + \eta_a c + \eta_i i + \mu) + c_1$

$s(t) = c_1 e^{-(\eta_a a \mp c + \eta_i i + \mu)t}, s(t) = s(0)e^{-(\eta_a a \mp c + \eta_i i + \mu)t} \geq 0.$

From Eq. (2),

$i' = \eta_a as + \eta_c cs + \eta_i is - (\gamma + \mu + \tau)i$

$i' \geq [\eta_i s - (\gamma + \mu + \tau)i]$

$lni \geq [\eta_i s - (\gamma + \mu + \tau)i]t + c_2,$

$i(t) = i(0) \times e^{[\eta_i s - (\gamma + \mu + \tau)i]t} \geq 0.$

Hence, the system (1-4) admits a positive solution.

Lemma 2: The solutions $(s, i, a, c) \varepsilon R_+^4$ of the system (1-4) are bounded at any time $t \geq 0$, and $\lim_{t \to \infty} SupN(t) \leq \frac{\Lambda}{\mu}$.

Proof. Consider the population function as follows:

$N = s + i.$

$\frac{dN}{dt} \leq \Lambda - \mu(s + i).$

$\frac{dN}{dt} + \mu N \leq \Lambda.$

$N(t) = Ae^{-\mu t} + \frac{\Lambda}{\mu}, SupN(t) \leq Ae^{-\mu t} + \frac{\Lambda}{\mu}$

$\lim_{t \to \infty} SupN(t) \leq \frac{\Lambda}{\mu}.$

Hence, the system (1-4) admits bounded solution and lie in the feasible region Z.

2.1 Model Equilibria

Considering the system (1-4) in which state variables treated as constant and the change in variables are assumed to be zero as follows:

$$0 = \Lambda - \eta_a as - \eta_c cs - \eta_i is - \mu s + \tau i, \tag{5}$$

$$0 = \eta_a as + \eta_c cs + \eta_i is - (\gamma + \mu + \tau)i \tag{6}$$

$$0 = -\alpha a + \beta c \tag{7}$$

$$0 = (\gamma + \mu)i - \delta c - kc \tag{8}$$

The system (5-8), admitted two types of equilibria that are anthrax free equilibrium (AFE) and anthrax existing equilibrium (AEE). The anthrax existing equilibrium (AEE) is denoted by $A_1 = (s^*, i^*, a^*, c^*)$

$$s^* = \frac{\gamma + \mu + \tau}{A^* + B^* + \eta_i}, i^* = \frac{\Lambda - \mu s^*}{A^* s^* + B^* s^* + \eta_i s^* - \tau}, a^* = \frac{\beta c^*}{\alpha}, c^* = \frac{(\gamma + \mu)i^*}{\delta + k}.$$

where $A^* = \eta_a \left(\frac{\beta}{\alpha}\right)\left(\frac{\gamma + \mu}{\delta + k}\right), B^* = \eta_c \left(\frac{\gamma + \mu}{\delta + k}\right)$.

And the anthrax free equilibrium (AFE) is denoted by $A_2 = (s^1, i^1, a^1, c^1) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$.

2.2 Reproduction Number

The reproduction number has a significant role in disease dynamics. It presented the ratio of infectivity in the population. For the sake, in the system of anthrax disease, there is only a single compartment is related to infected ness. Without loss of generality, we consider

$$\frac{di}{dt} > 0$$

$$\eta_a as + \eta_c cs + \eta_i is - (\gamma + \mu + \tau)i > 0$$

By assuming the anthrax free equilibrium $A_2 = (s^1, i^1, a^1, c^1) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ as follows:

$\frac{\Lambda \eta_i}{\mu} > (\gamma + \mu + \tau), \frac{\eta_i \Lambda}{\mu(\gamma + \mu + \tau)} > 1.$

$R_0 = \frac{\eta_i \Lambda}{\mu(\gamma + \mu + \tau)} > 1.$

Note that, R_0 is called the reproduction number of the model.

2.3 Local Stability

In this section, we discussed two well-known theorems for stability. Considering the system (5-8) as functions of the equations.

$$F_1 = \Lambda - \eta_a as - \eta_c cs - \eta_i is - \mu s + \tau i, \quad F_2 = \eta_a as + \eta_c cs + \eta_i is - (\gamma + \mu + \tau)i, \quad F_3 = -\alpha a + \beta c,$$
$$F_4 = (\gamma + \mu)i - \delta c - kc.$$

The partial derivatives along with the states variables as follows:

where $\frac{\partial F_4}{\partial s} = 0$, $\frac{\partial F_3}{\partial s} = 0$, $\frac{\partial F_2}{\partial s} = \eta_a a + \eta_c c + \eta_i i$, $\frac{\partial F_1}{\partial s} = -\eta_a a - \eta_c c - \eta_i i - \mu$, $\frac{\partial F_1}{\partial i} = -\eta_i s + \tau$, $\frac{\partial F_2}{\partial i} = \eta_i is - (\gamma + \mu + \tau)$, $\frac{\partial F_3}{\partial i} = 0$, $\frac{\partial F_4}{\partial i} = (\gamma + \mu)$, $\frac{\partial F_1}{\partial a} = -\eta_a s$, $\frac{\partial F_2}{\partial a} = \eta_a s$, $\frac{\partial F_3}{\partial a} = -\alpha$, $\frac{\partial F_4}{\partial a} = 0$, $\frac{\partial F_1}{\partial c} = -\eta_c s$, $\frac{\partial F_2}{\partial c} = \eta_c s$, $\frac{\partial F_3}{\partial c} = \beta$, $\frac{\partial F_4}{\partial c} = -\delta - k$.

The general form of the Jacobin matrix is defined as

$$J = \begin{bmatrix} \dfrac{\partial F_1}{\partial s} & \dfrac{\partial F_1}{\partial i} & \dfrac{\partial F_1}{\partial a} & \dfrac{\partial F_1}{\partial c} \\[2mm] \dfrac{\partial F_2}{\partial s} & \dfrac{\partial F_2}{\partial i} & \dfrac{\partial F_2}{\partial a} & \dfrac{\partial F_2}{\partial c} \\[2mm] \dfrac{\partial F_3}{\partial s} & \dfrac{\partial F_3}{\partial i} & \dfrac{\partial F_3}{\partial a} & \dfrac{\partial F_3}{\partial c} \\[2mm] \dfrac{\partial F_4}{\partial s} & \dfrac{\partial F_4}{\partial i} & \dfrac{\partial F_4}{\partial a} & \dfrac{\partial F_4}{\partial c} \end{bmatrix}$$

$$J = \begin{bmatrix} -\eta_a a - \eta_c c - \eta_i i - \mu & -\eta_i s + \tau & -\eta_a s & -\eta_c s \\ \eta_a a + \eta_c c + \eta_i i & \eta_i is - (\gamma + \mu + \tau) & \eta_a s & \eta_c s \\ 0 & 0 & -\alpha & \beta \\ 0 & (\gamma + \mu) & 0 & -\delta - k \end{bmatrix} \tag{9}$$

Theorem 1: If $R_0 < 1$, then $A_2 = (s^1, i^1, a^1, c^1) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ is locally asymptotically stable in the system (1-4). Otherwise, unstable.

Proof. The Jacobean matrix of Eq. (9) at A_2 is as follows:

$$J(A_2) = \begin{bmatrix} -\mu & -\eta_i\left(\frac{\Lambda}{\mu}\right) + \tau & -\eta_a\left(\frac{\Lambda}{\mu}\right) & -\eta_c\left(\frac{\Lambda}{\mu}\right) \\ 0 & \eta_i\left(\frac{\Lambda}{\mu}\right) - (\gamma + \mu + \tau) & \eta_a\left(\frac{\Lambda}{\mu}\right) & \eta_c\left(\frac{\Lambda}{\mu}\right) \\ 0 & 0 & -\alpha & \beta \\ 0 & (\gamma + \mu) & 0 & -\delta - k \end{bmatrix}$$

$$|J(A_2) - \lambda I| = \begin{vmatrix} -\mu - \lambda & -\eta_i\left(\frac{\Lambda}{\mu}\right) + \tau & -\eta_a\left(\frac{\Lambda}{\mu}\right) & -\eta_c\left(\frac{\Lambda}{\mu}\right) \\ 0 & \eta_i\left(\frac{\Lambda}{\mu}\right) - (\gamma + \mu + \tau) - \lambda & \eta_a\left(\frac{\Lambda}{\mu}\right) & \eta_c\left(\frac{\Lambda}{\mu}\right) \\ 0 & 0 & -\alpha - \lambda & \beta \\ 0 & (\gamma + \mu) & 0 & -\delta - K - \lambda \end{vmatrix} = 0.$$

$$(-\mu - \lambda)\begin{vmatrix} \eta_i\left(\frac{\Lambda}{\mu}\right) - (\gamma + \mu + \tau) - \lambda & \eta_a\left(\frac{\Lambda}{\mu}\right) & \eta_c\left(\frac{\Lambda}{\mu}\right) \\ 0 & -\alpha - \lambda & \beta \\ \gamma + \mu & 0 & -\delta - k - \lambda \end{vmatrix} = 0.$$

$$\lambda_1 = -\mu < 0,$$

$$\left(\eta_i\left(\frac{\Lambda}{\mu}\right) - (\gamma + \mu + \tau) - \lambda\right)\begin{vmatrix} -\alpha - \lambda & \beta \\ 0 & -\delta - k - \lambda \end{vmatrix} + (\gamma + \mu)(-1)^{3+1}\begin{vmatrix} \eta_a\left(\frac{\Lambda}{\mu}\right) & \eta_c\left(\frac{\Lambda}{\mu}\right) \\ -\alpha - \lambda & \beta \end{vmatrix} = 0$$

$$\left(\eta_i\frac{\Lambda}{\mu} - (\gamma + \mu + i) - \lambda\right)(\alpha\delta + \alpha k + \alpha\lambda + \delta\lambda + k\lambda + \lambda^2) + (\gamma + \mu)\left(\eta\frac{\Lambda}{\mu}\beta + \alpha\eta_c\frac{\Lambda}{\mu} + \lambda\eta_c\frac{\Lambda}{\mu}\right) = 0$$

$$(A - \lambda)(B + C\lambda + \lambda^2) + D + E\lambda = 0.$$

where $A = \eta_i\frac{\Lambda}{\mu} - (\gamma + \mu + i)$, $B = \alpha\delta + \alpha k$, $C = \delta + k$, $D = (\gamma + \mu)\left(\eta\frac{\Lambda}{\mu}\beta + \alpha\eta_c\frac{\Lambda}{\mu}\right)$, $E = (\gamma + \mu)\eta_c\frac{\Lambda}{\mu}$.

$$\lambda^3 - (A - C)\lambda^2 - (AC - B + E)\lambda - (AB + D) = 0.$$

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0.$$

Where $a_2 = A - C$, $a_1 = E - AC + B$, $a_0 = -(AB + D)$.

By using the Routh Hurwitz criterion of 3rd order, the following constraint is satisfied $a_2, a_0 > 0$, and $a_2 a_1 > a_0$ when $R_0 < 1$. Hence the anthrax-free equilibrium A_2 is locally asymptotically stable (LAS).

Theorem 2: If $R_0 > 1$, then $A_1 = (s^*, i^*, a^*, c^*)$ is locally asymptotically stable in the system (1-4). Otherwise, unstable.

Proof: The Jacobean matrix Eq. (9) at A_1 is as follows:

$$J(A_1) = \begin{bmatrix} -\eta_a a^* - \eta_c c^* - \eta_i i^* - \mu & -\eta_i s^* + \tau & -\eta_a s^* & -\eta_c s^* \\ \eta_a a^* + \eta_c c^* + \eta_i i^* & \eta_i s^* - (\gamma + \mu + \tau) & \eta_a s^* & \eta_c s^* \\ 0 & 0 & -\alpha & \beta \\ 0 & (\gamma + \mu) & 0 & -\delta - k \end{bmatrix}$$

$$|J(A_1) - \lambda I| = \begin{bmatrix} -\eta_a a^* - \eta_c c^* - \eta_i i^* - \mu - \lambda & -\eta_i s^* + \tau & -\eta_a s^* & -\eta_c s^* \\ \eta_a a^* + \eta_c c^* + \eta_i i^* & \eta_i s^* - (\gamma + \mu + \tau) - \lambda & \eta_a s^* & \eta_c s^* \\ 0 & 0 & -\alpha - \lambda & \beta \\ 0 & (\gamma + \mu) & 0 & -\delta - k - \lambda \end{bmatrix} = 0.$$

$$(-\eta_a a^* - \eta_c c^* - \eta_i i^* - \mu - \lambda) \begin{vmatrix} \eta_i s^* - (\gamma + \mu + \tau) - \lambda & \eta_a s^* & \eta_c s^* \\ 0 & -\alpha - \lambda & \beta \\ r + \mu & 0 & -\delta - k - \lambda \end{vmatrix}$$

$$+ (-1)^{2+1} (\eta_a a^* + \eta_c^* + \eta_i i^*) \begin{vmatrix} -\eta_i s^* + \tau & \eta_a s^* & -\eta_c s^* \\ 0 & -\alpha - \lambda & \beta \\ \gamma + \mu & 0 & -\delta - k - \lambda \end{vmatrix} = 0$$

$$(g - \lambda) \begin{vmatrix} g_1 - \lambda & g_2 & g_3 \\ 0 & -\alpha - \lambda & \beta \\ g_4 & 0 & g_5 - \lambda \end{vmatrix} - h \begin{vmatrix} h_1 & h_2 & h_3 \\ 0 & -\alpha - \lambda & \beta \\ h_4 & 0 & h_5 - \lambda \end{vmatrix} = 0$$

where $g = -\eta_a a^* - \eta_c c^* - \eta_i i^* - \mu$, $g_1 = \eta_i s^* - (\gamma + \mu + \tau)$, $g_2 = \eta_a s^*$, $g_3 = \eta_c s^*$, $g_4 = r + \mu$, $g_5 = -\delta - k$, $h = \eta_a a^* + \eta_c c^* + \eta_i i^*$, $h_1 = -\eta_i s^* + \tau$, $h_2 = \eta_a s^*$, $h_3 = -\eta_c s^*$, $h_4 = r + \mu$, $h_5 = -\delta - k$.

$$(g - \lambda)\{(g_1 - \lambda)\{(\alpha + \lambda)(-g_5 + \lambda) - 0\} + g_4(g_2\beta + g_3(\alpha + \lambda))\} - h\{h_1((-\alpha - \lambda)(h_5 - \lambda) - 0)$$

$$+ h_4(h_2\beta + h_3(\alpha + \lambda))\} = 0$$

$$\Rightarrow (g - \lambda)\{(G_1) + (G_2)\lambda + (G_3)\lambda^2 - \lambda^3\} - h\{(H_1) + (H_2)\lambda + h_1\lambda^2\} = 0$$

where $G_1 = \alpha g_3 g_4 - \alpha g_1 g_5 + \beta g_2 g_4$, $G_2 = \alpha g_1 + \alpha g_5 + g_3 g_4 - g_1 g_5$, $G_3 = -\alpha + g_1 + g_5$, $H_1 = \alpha h_3 h_4 - \alpha h_1 h_5 + \beta h_2 h_4$, $H_2 = \alpha h_1 + h_3 h_4 - h_1 h_5$,

$$g G_1 + g G_2 \lambda + g G_3 \lambda^2 - g\lambda^3 - G_1\lambda - G_2\lambda^2 - G_3\lambda^3 + \lambda^4 - hH_1 - hH_2\lambda - hh_1\lambda^2 = 0.$$

$$\lambda^4 + (-g - G_3)\lambda^3 + (gG_3 - G_2 - hh_1)\lambda^2 + (gG_2 - G_1 - hH_2)\lambda + (gG_1 - hH_1) = 0.$$

$$\lambda^4 + b_0\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0.$$

where $b_0 = (-g - G_3)$, $b_1 = gG_3 - G_2 - hh_1$, $b_2 = gG_2 - G_1 - hH_2$, $b_3 = gG_1 - hH_1$,

By using Routh Hurwitz criteria of order 4th, all constraints have been satisfied. So, anthrax existing equilibrium A_1 is locally asymptotically stable (LAS).

3 Computer Methods

In this section, we discussed some well-known computer methods like Euler, Runge Kutta, and the non-standard finite difference method for the system (1-4).

3.1 Euler Method

This method could be applied in the system (1-4) as follows:

$$s^{n+1} = s^n + h[\Lambda - \eta_a a^n s^n - \eta_c c^n s^n - \eta_i i^n s^n - \mu s^n + \tau i^n]. \tag{10}$$

$$i^{n+1} = i^n + h[\eta_a a^n s^n + \eta_c c^n s^n + \eta_i i^n s^n - (\gamma + \mu + \tau)i^n]. \tag{11}$$

$$a^{n+1} = a^n + h[-\alpha a^n + \beta c^n]. \tag{12}$$

$$c^{n+1} = c^n + h[(\gamma + \mu)i^n - (\delta + K)c^n]. \tag{13}$$

where n = 0,1,2,3… and discretization gap is denoted by h.

3.2 Runge Kutta Method

This method could be applied in the system (1-4) as follows:

Stage 1

$$K_1 = h[\Lambda - \eta_a a^n s^n - \eta_c c^n s^n - \eta_i i^n s^n - \mu s^n + \tau i^n].$$
$$L_1 = h[\eta_a a^n s^n + \eta_c c^n s^n + \eta_i i^n s^n - (\gamma + \mu + \tau)i^n].$$
$$M_1 = h[-\alpha a^n + \beta c^n].$$
$$N_1 = h[(\gamma + \mu)i^n - (\delta + K)c^n].$$

Stage 2

$$K_2 = h\left[\Lambda - \eta_a\left(a^n + \frac{M_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) - \eta_c\left(c^n + \frac{N_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) - \eta_i\left(i^n + \frac{L_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) - \mu\left(s^n + \frac{K_1}{2}\right) + \tau\left(i^n + \frac{L_1}{2}\right)\right].$$
$$L_2 = h\left[\eta_a\left(a^n + \frac{M_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) + \eta_c\left(c^n + \frac{N_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) + \eta_i\left(i^n + \frac{L_1}{2}\right)\left(s^n + \frac{K_1}{2}\right) - (\gamma + \mu + \tau)\left(i^n + \frac{L_1}{2}\right)\right].$$
$$M_2 = h\left[-\alpha\left(a^n + \frac{M_1}{2}\right) + \beta\left(c^n + \frac{N_1}{2}\right)\right].$$
$$N_2 = h\left[(\gamma + \mu)\left(i^n + \frac{L_1}{2}\right) - (\delta + K)\left(c^n + \frac{N_1}{2}\right)\right].$$

Stage 3

$$K_3 = h\left[\Lambda - \eta_a\left(a^n + \frac{M_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) - \eta_c\left(c^n + \frac{N_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) - \eta_i\left(i^n + \frac{L_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) - \mu\left(s^n + \frac{K_2}{2}\right) + \tau\left(i^n + \frac{L_2}{2}\right)\right].$$
$$L_3 = h\left[\eta_a\left(a^n + \frac{M_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) + \eta_c\left(c^n + \frac{N_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) + \eta_i\left(i^n + \frac{L_2}{2}\right)\left(s^n + \frac{K_2}{2}\right) - (\gamma + \mu + \tau)\left(i^n + \frac{L_2}{2}\right)\right].$$
$$M_3 = h\left[-\alpha\left(a^n + \frac{M_2}{2}\right) + \beta\left(c^n + \frac{N_2}{2}\right)\right].$$
$$N_3 = h\left[(\gamma + \mu)\left(i^n + \frac{L_2}{2}\right) - (\delta + K)\left(c^n + \frac{N_2}{2}\right)\right].$$

Stage 4

$$K_4 = h[\Lambda - \eta_a(a^n + M_1)(s^n + K_1) - \eta_c(c^n + N_1)(s^n + K_1) - \eta_i((i^n + L_1)(s^n + K_1) + K_1 - \mu(s^n + K_1) + \tau(c^n + N_1)].$$
$$L_2 = h[\eta_a(a^n + M_1)(s^n + K_1) + \eta_c(c^n + N_1)(s^n + K_1) + \eta_i(i^n + L_1)(s^n + K_1) - (\gamma + \mu + \tau)(i^n + L_1)].$$
$$M_2 = h[-\alpha(a^n + M_1) + \beta(c^n + N_1)].$$
$$N_2 = h[(\gamma + \mu)(i^n + L_1) - (\delta + K)(c^n + N_1)].$$

Final stage

$$
\left.\begin{array}{l}
s^{n+1} = s^n + \frac{1}{6}[K_1 + 2k_2 + 2k_3 + k_4] \\
i^{n+1} = i^n + \frac{1}{6}[L_1 + 2L_2 + 2L_3 + L_4] \\
a^{n+1} = a^n + \frac{1}{6}[M_1 + 2M_2 + 2M_3 + M_4] \\
c^{n+1} = c^n + \frac{1}{6}[N_1 + 2N_2 + 2N_3 + N_4]
\end{array}\right\}
\tag{14}
$$

where h is any discretization and $n \geq 0$.

3.3 Non-Standard Finite Difference Method

This method could be applied in the system (1-4) as follows:

$$
s^{n+1} = \frac{s^n + h(\Lambda + \tau i^n)}{1 + h\eta_a a^n + \eta_c hc^n + h\eta_i i^n + h\mu}.
\tag{15}
$$

$$
i^{n+1} = \frac{i^n + h\eta_a s^n a^n + \eta_c s^n hc^n + h\eta_i i^n s^n + h\mu}{1 + h(\gamma + \mu + \tau)}.
\tag{16}
$$

$$
a^{n+1} = \frac{a^n + h\beta c^n}{1 + \alpha h}.
\tag{17}
$$

$$
c^{n+1} = \frac{c^n + h(\gamma + \mu)i^n}{1 + h(\delta + k)}.
\tag{18}
$$

where h is any discretization and $n \geq 0$.

3.4 Convergence Analysis

Theorem 3: For any $n \geq 0$, the proposed NSFD method is stable if the Eigenvalues of the system lie in the unit circle if $R_0 < 1$.

Proof. Consider the right-hand sides of the system (15-18), as function W, X, Y, and Z as follows:

$$
W = \frac{s + h(\Lambda + \tau i)}{1 + h\eta_a a + \eta_c hc + h\eta_i i + h\mu}, \quad X = \frac{i + h\eta_a sa + \eta_c shc + h\eta_i is + h\mu}{1 + h(\gamma + \mu + \tau)}, \quad Y = \frac{a + h\beta c}{1 + \alpha h}, \quad Z = \frac{c + h(\gamma + \mu)i}{1 + h(\delta + k)}.
$$

The partial derivatives with respect to the state variables as follows:

where $\quad \dfrac{\partial W}{\partial s} = \dfrac{1}{1 + h\mu}, \quad \dfrac{\partial W}{\partial i} = \dfrac{h\tau}{1 + h\mu} - \dfrac{h\eta_i(\frac{\Lambda}{\mu} + h\Lambda)}{(1 + h\mu)^2}, \quad \dfrac{\partial W}{\partial a} = -\dfrac{h\eta_a(\frac{\Lambda}{\mu} + h\Lambda)}{(1 + h\mu)^2}, \quad \dfrac{\partial W}{\partial c} = -\dfrac{h\eta_c(\frac{\Lambda}{\mu} + h\Lambda)}{(1 + h\mu)^2},$

$\dfrac{\partial X}{\partial s} = 0, \quad \dfrac{\partial X}{\partial i} = \dfrac{1 + h\eta_i(\frac{\Lambda}{\mu})}{1 + h(\gamma + \mu + \tau)}, \quad \dfrac{\partial X}{\partial a} = \dfrac{h\eta_a(\frac{\Lambda}{\mu})}{1 + h(\gamma + \mu + \tau)}, \quad \dfrac{\partial X}{\partial c} = \dfrac{h\eta_c(\frac{\Lambda}{\mu})}{1 + h(\gamma + \mu + \tau)}, \quad \dfrac{\partial Y}{\partial s} = 0, \quad \dfrac{\partial Y}{\partial i} = 0,$

$\dfrac{\partial Y}{\partial a} = \dfrac{1}{1 + h\alpha}, \quad \dfrac{\partial Y}{\partial c} = \dfrac{h\beta}{1 + h\alpha}, \quad \dfrac{\partial Z}{\partial s} = 0, \quad \dfrac{\partial Z}{\partial i} = \dfrac{h(\gamma + \mu)}{1 + h(\delta + k)}, \quad \dfrac{\partial Z}{\partial a} = 0, \quad \dfrac{\partial Z}{\partial c} = \dfrac{1}{1 + h(\delta + k)}.$

The Jacobean matrix at anthrax free equilibrium is as follows:

$$J\left(\frac{\Delta}{\mu},0,0,0\right) = \begin{bmatrix} \dfrac{1}{1+h\mu} & \dfrac{h\tau}{1+h\mu} - \dfrac{h\eta_i(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} & -\dfrac{h\eta_a(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} & -\dfrac{h\eta_c(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} \\[2ex] 0 & \dfrac{1+h\eta_i(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} & \dfrac{h\eta_a(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} & \dfrac{h\eta_c(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} \\[2ex] 0 & \text{o} & \dfrac{1}{1+h\alpha} & \dfrac{h\beta}{1+h\alpha} \\[2ex] 0 & \dfrac{h(\gamma+\mu)}{1+h(\delta+k)} & 0 & \dfrac{1}{1+h(\delta+k)} \end{bmatrix}.$$

$$|J-\lambda I| = \begin{vmatrix} \dfrac{1}{1+h\mu}-\lambda & \dfrac{h\tau}{1+h\mu} - \dfrac{h\eta_i(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} & -\dfrac{h\eta_a(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} & -\dfrac{h\eta_c(\frac{\Delta}{\mu}+h\Lambda)}{(1+h\mu)^2} \\[2ex] 0 & \dfrac{1+h\eta_i(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)}-\lambda & \dfrac{h\eta_a(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} & \dfrac{h\eta_c(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} \\[2ex] 0 & \text{o} & \dfrac{1}{1+h\alpha}-\lambda & \dfrac{h\beta}{1+h\alpha} \\[2ex] 0 & \dfrac{h(\gamma+\mu)}{1+h(\delta+k)} & 0 & \dfrac{1}{1+h(\delta+k)}-\lambda \end{vmatrix} = 0.$$

$$\lambda_1 = \left|\frac{1}{1+h\mu}\right| < 1 \ ,$$

$$\begin{vmatrix} \dfrac{1+h\eta_i(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)}-\lambda & \dfrac{h\eta_a(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} & \dfrac{h\eta_c(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)} \\[2ex] \text{o} & \dfrac{1}{1+h\alpha}-\lambda & \dfrac{h\beta}{1+h\alpha} \\[2ex] \dfrac{h(\gamma+\mu)}{1+h(\delta+k)} & 0 & \dfrac{1}{1+h(\delta+k)}-\lambda \end{vmatrix} = 0$$

$$\begin{vmatrix} h_1-\lambda & h_2 & h_3 \\ \text{o} & h_4-\lambda & h_5 \\ h_6 & 0 & h_7-\lambda \end{vmatrix} = 0$$

where $h_1 = \dfrac{1+h\eta_i(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)}$, $h_2 = \dfrac{h\eta_a(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)}$, $h_3 = \dfrac{h\eta_c(\frac{\Delta}{\mu})}{1+h(\gamma+\mu+\tau)}$, $h_4 = \dfrac{1}{1+h\alpha}$, $h_5 = \dfrac{h\beta}{1+h\alpha}$,

$h_6 = \dfrac{h(\gamma+\mu)}{1+h(\delta+k)}$, $h_7 = \dfrac{1}{1+h(\delta+k)}$.

$\lambda^3 + h_8\lambda^2 + h_9\lambda + h_{10} = 0$.

where $h_8 = -(h_1 + h_4 + h_7)$, $h_9 = -(-h_1 h_4 - h_1 h_7 - h_4 h_7 + h_3 h_6)$, $h_{10} = -(h_1 h_4 h_7 - h_1 h_2 h_5 - h_3 h_4 h_6)$.

By using the Mathematica, this is guarantee to the fact that all values of Jacobian lie in a unit circle, as desired.

3.5 Computer Results

In this section, we used the scientific literature presented in Tab. 2, for the simulating behavior of the system (5-8) at both equilibria of the model as follows:

Table 2: Value of parameters

Parameters	Values
Λ	0.05
δ	0.025
α	0.035
β	0.035
η_i	0.001 (AFE) 1.001 (AEE)
τ	0.0001
γ	0.02
μ	0.05
k	0.1
η_a	0.005

4 Results and Concluding Remarks

In Figs. 2a and 2b, we used the command-built software ODE-45 to simulate the behavior of the model at any time t. In Figs. 3a and 3b, the behavior of the computer method like Euler at different time step sizes are presented in which we can analyze the said method is converges for the small-time step size. In Figs. 4a and 4b, the second important method like Runge Kutta is also diverged and depends upon time step size. In meanwhile, Figs. 5a and 5b, is the true sense results simulates by non-standard finite difference method at any time step size. This computer method has the advantage over the other two methods like Euler and Runge Kutta. Figs. 6a and 6d is the ethnicity of the proposed computer method. Independent of time step size, low-cost and effective technique. Through the study, anthrax disease modeling is analyzed along with analytical approaches and computer techniques. The model is based on four compartments two humans (Susceptible and infected) and two related to dead bodies and spores' germs (bacteria). The human compartment along with positivity, boundedness, equilibria, reproduction number, and local stability is studied rigorously. In the future, we could extend this type of modeling to other complex epidemiological models and their branches.
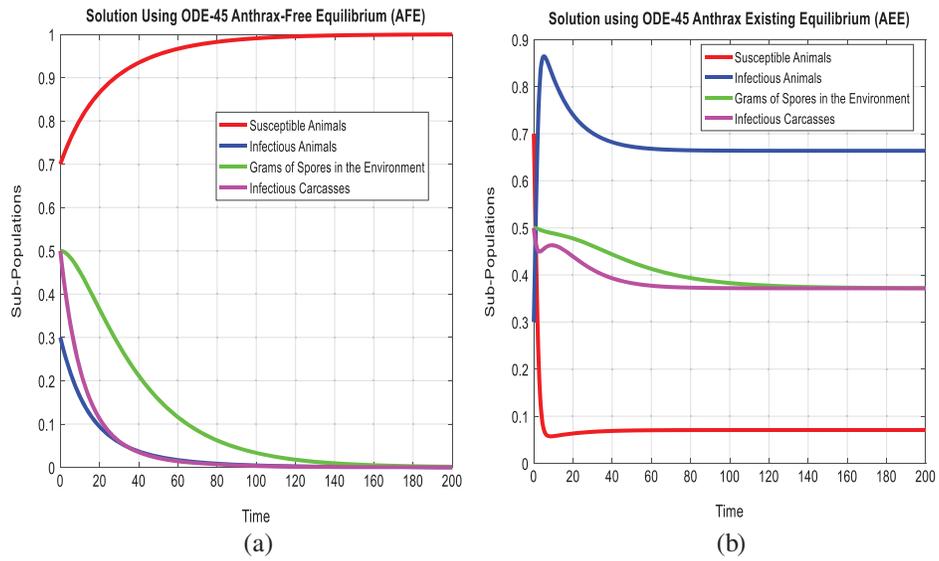
Figure 2: Combined graphical behavior for the equilibria and converges at any time t (a) sub-populations at anthrax-free equilibrium (b) Sub-populations at anthrax existing equilibrium
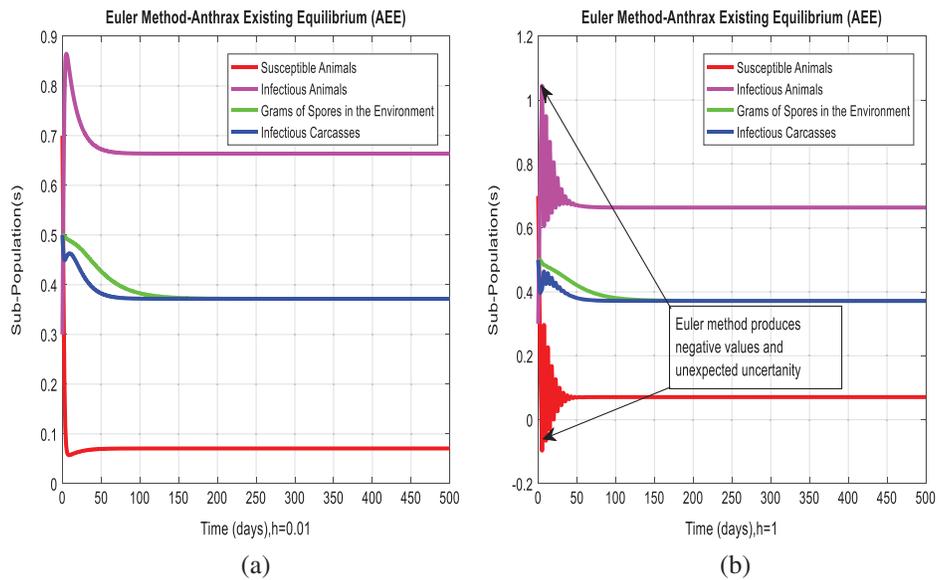
Figure 3: Combined graphical behavior for the equilibria of the model (a) Euler method for sub-populations at anthrax existing equilibrium when h = 0.01 (b) Euler method for sub-populations at anthrax existing equilibrium when h = 1
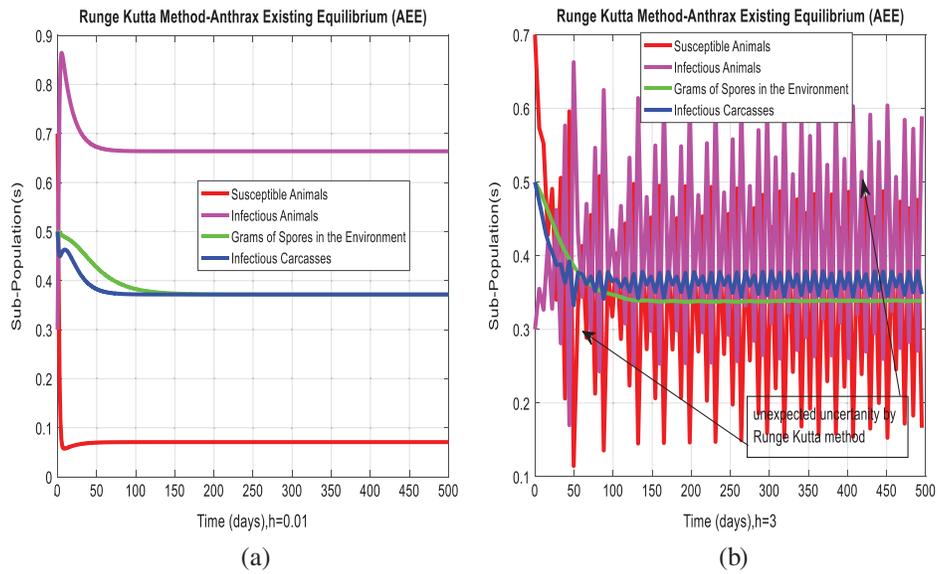
Figure 4: Combined graphical behavior for the equilibria of the model (a) Runge Kutta method for sub-populations at anthrax existing equilibrium when h = 0.01 (b) Runge Kutta method for sub-populations at anthrax existing equilibrium when h = 3
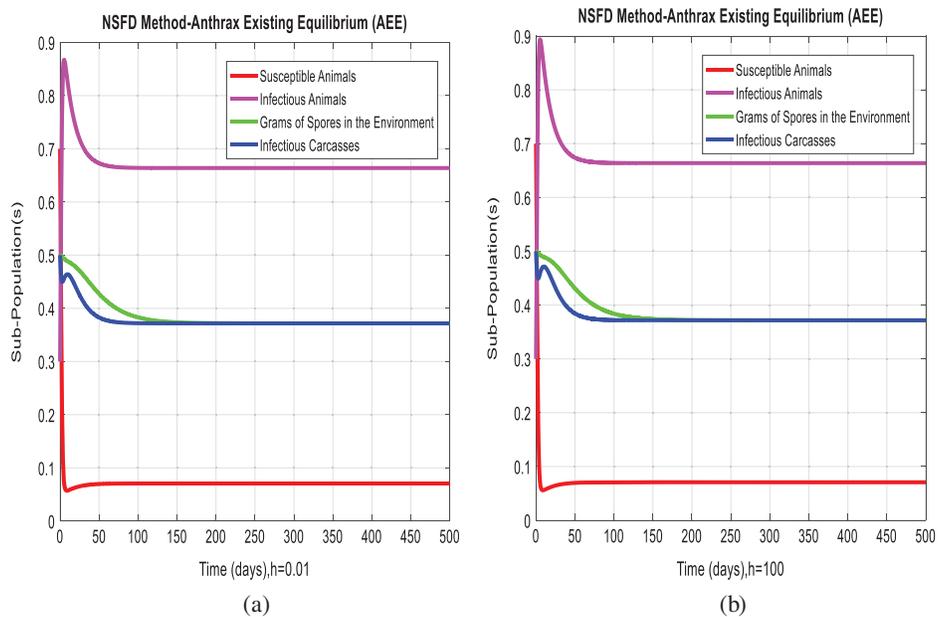
Figure 5: Combined graphical behavior for the equilibria of the model (a) NSFD method for sub-populations at anthrax existing equilibrium when h = 0.01 (b) NSFD method for sub-populations at anthrax existing equilibrium when h = 100
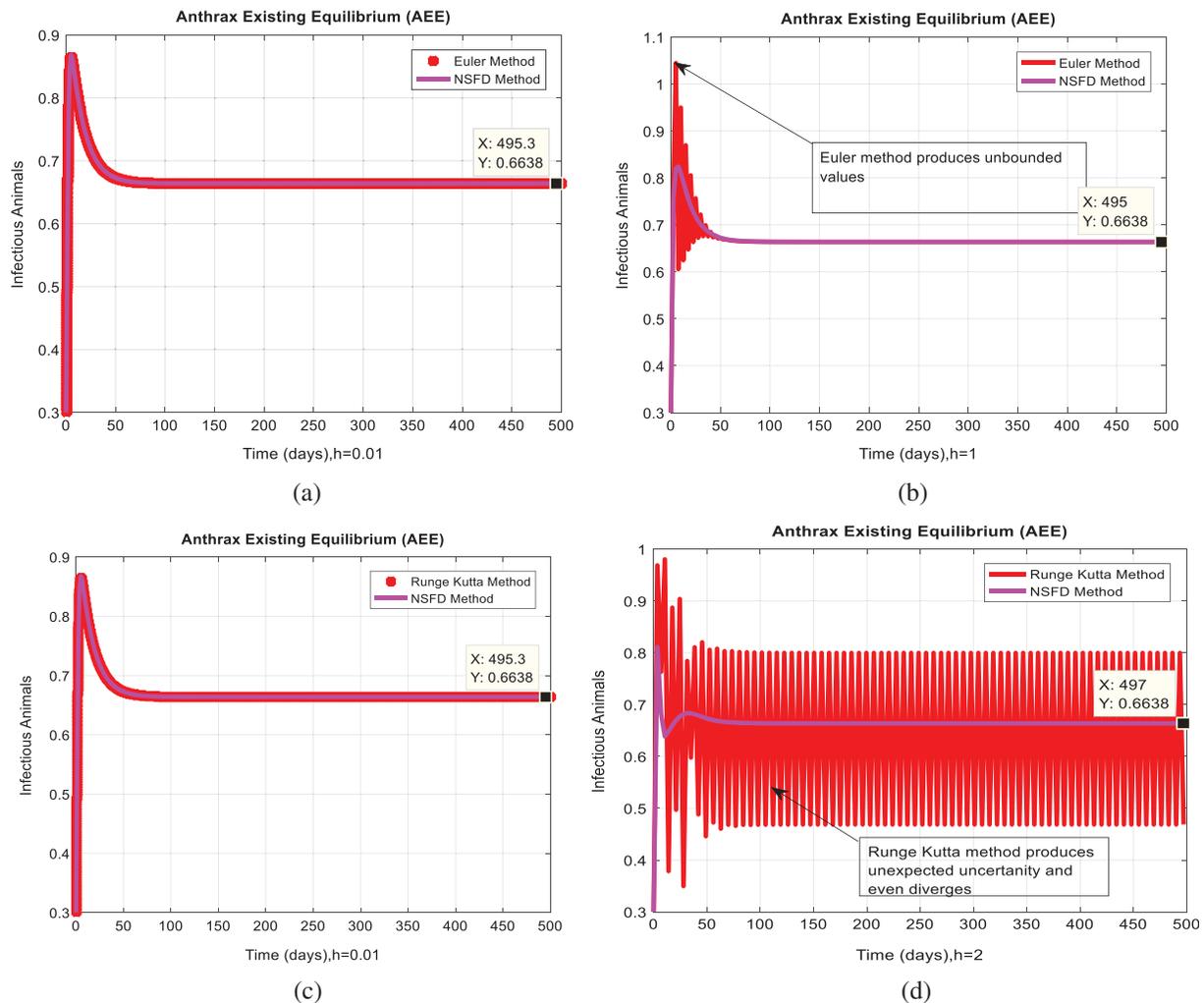
Figure 6: Combined behaviors of NSFD with Euler and Runge Kutta at different step sizes (a) Converges to true equilibria of NSFD and Euler methods for the infected population at anthrax existing equilibrium when h = 0.01 (b) Euler method diverges and produce negativity at h = 1 (c) Converges to true equilibria of NSFD and Runge Kutta methods for the infected population at anthrax existing equilibrium when h = 0.01 (d) Runge Kutta method diverges, produce negativity, and even fluctuation at h = 2

Conflicts of Interest: The authors declare that they have no conflicts of interest to report regarding the present study.

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