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A STUDY OF FRACTIONAL BOVINE TUBERCULOSIS MODEL WITH VACCINATION ON HUMAN POPULATION

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Abstract. A bacterial and zoonotic disease called bovine tuberculosis (bTB) can be contracted by breathing in aerosols, consuming unpasteurized milk, or eating raw meat. The evolution of bovine tuberculosis transmission in both human and animal populations is investigated in this research using a fractional order model with caputo sensing and a compartment for human vaccination. The threshold quantity R_0 was also constructed using Volterra-type Lyapunov functions, LaSalle's invariance principle, and the Routh-Hurwitz criterion to identify the sick state and provide conditions that guarantee the local and global asymptotic stability of the equilibria. In order to determine the variables that control the dynamics of bTB, we performed a sensitivity study. The analysis indicates that factors influencing the spread of bTB include the rate of environmental contamination, the rate of bTB transmission from animal to animal, and the rate at which bTB is contracted by people from infected animals and the environment. However, the disease becomes less common in humans as vaccination rates rise and consumption of the contaminated environment's products (meat and dairy products) declines. For the management of bTB, it is recommended to implement educational initiatives, monitor the environment, treat affected individuals, administer

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immunizations, and confine contaminated animals. Numerical experiments are used to show how useful the found theoretical results are.

Keywords: fractional order model; zoonotic disease; bovine tuberculosis; vaccination compartiment.

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1. INTRODUCTION

In recent years, Africa has made strides in the fight against tuberculosis (TB), but numerous challenges still stand in the way of efforts to eradicate this avoidable and treatable illness. Global efforts to eradicate the illness by 2030 appear to be lagging behind schedule at the present time [1, 2]. Tuberculosis (TB) is a chronic infectious illness that mostly affects the respiratory system. Africa had the highest cases, followed by India, China, and Indonesia in order of prevalence, with 72%, 27%, 9%, and 8%, respectively, according to studies in [3].
COVID-19 has an effect on both TB research and the relationship between TB and care. The reallocation of resources to the COVID-19 response has made it more difficult for numerous countries to provide essential services. Many people with tuberculosis have had trouble getting treatment because of the lockdowns. COVID-19 has an impact on the ability to identify drug-resistant tuberculosis, according to World Health. In 2020, there were 28% fewer cases recorded in the WHO's African Region than there were in 2019[1].

Bovine tuberculosis is a zoonotic infectious disease that the OIE (Office International des Epizooties) designates as a class *B* animal pandemic. Infected animals can be the main source of infection for both humans and other animals. The main pathways of transmission are the gut and respiratory systems. Healthy people and animals can become infected by sick animals by coming into contact with them or drinking their raw milk, [4], [5]. The disease has a major negative economic impact due to the slaughter of bTB-infected animals when they become ill. [5]. Furthermore, bTB has a negative impact on people's health, which can occasionally result in fatalities [6]. It may lead to the loss of their self-employment for some employees, particularly those who depend on raising cattle as their main source of income [7]. Inhaling aerosols, consuming raw meat, and drinking unpasteurized milk are the three main ways that bovine tuberculosis spreads from animals to humans. Additional methods that bTB spreads

among animals include intimate contact between infected and uninfected animals, consumption of contaminated milk, particularly during lactation, and inhalation of aerosols[8] [9].

The most well-known and often employed method for diagnosing bTB is the intradermal skin test [10]. According to numerous articles, its main shortcomings are its varying sensitivity and specificity. Additionally, tuberculosis vaccination techniques hinder this test since sensitized animals produce false-positive results [9]. A deterministic mathematical model is developed in [5] to investigate the dynamics of bTB transmission in people and animals living in contaminated environments. The fundamental reproduction number R_0 is determined to ascertain the disease's behavior.According to the sensitivity analysis, the rate of production of dairy products, the rate of bTB transmission from animal to animal, and the rate at which humans contract bTB from infected dairy products and animals are what propel bTB transmission.

An intriguing article [11] evaluates the effects of the BCG vaccination on cattle and is based on a meta-analysis by experts from Ethiopia, the Netherlands, the United States, the United Kingdom, and India. In endemic locations, BCG vaccination may speed the control of bTB, according their findings. The immunology of Mycobacterium bovis (Mb) infection has been covered in some papers. Lung and lymph node lesions, which ultimately lead to the formation of granulomas, define the pathophysiology of bovine TB. The chronic development and immunopathology of bTB have many characteristics with those of human TB, according to a new study by Blanco [9]. Ahmad, Khan, Ahmad, Stanimirovic, and Chu in [12] created the reaction-diffusion model and used the fractional differential equation to derive standard solutions to the nonlinear partial differential equation. The fractional differential equation, which may be used in a variety of contexts, is an effective tool for comprehending the dynamics of diverse life events in fractional order.

In [13], differential equations of integer and fractional orders are used to build mathematical models for the dynamics of Potato Leaf Roll Virus propagation. The models considered both the Potato and Vector populations. The potato leaf roll virus (PLRV) model was initially proposed in integer order, and it was then extended into fractional order since fractional order provides memory and other benefits for replicating actual events.

Review of fractional epidemic models is the title of a publication by Chen et al[14]. that focuses on reviewing various fractional epidemic model types and evaluating the results of epidemiological modeling, particularly the fractional epidemic model. To address fractional epidemic models, they created straightforward and efficient analytical procedures that may be readily expanded and applied to other fractional models. These methods can help the concerned organizations stop, manage, and even predict infectious disease epidemics.

To the authors' knowledge, no studies have been conducted to model the transmission of bovine TB using classes for vaccination and contaminated environment. Therefore, this paper created a fractional-order mathematical model of bovine TB by accounting for vaccination and a contaminated environment. According to the findings, lowering the infection rate σ_A and contact rate σ_H significantly aids in the management of the TB disease in animal human population respectively. Additionally, disinfecting by warming the dairy products and cooking very well the meat has a significant positive impact on the disease's control. This is because it increases the elimination rate of contaminated environment ω.

This paper is organized as follows: In Section 2, the formulation and outline of the suggested model are presented. Section 3's primary objective is the model's analysis. Section 4 covers the numerical simulation of the model. Section 5 concludes with a summary and recommendations.

2. Model Description and Formulation

According to their disease condition in the system, the model separates the overall human and animal populations into seven (7) sub-populations (compartments) at any given time (t), and another compartment for the contaminated environment C_e.

We have the following assumptions:

(1) It is assumed that birth rates and immigration rates into the susceptible human population are stable.

(2) The direct transmission between people, between people and animal, and between animals follows the usual occurrence.

(3) The model does not have a recovery class because it is presumed that there is no natural recovery.

(4) It is believed that after contracting bTB, people or animals take some time before developing clinical symptoms.

(5) Humans can catch the disease by consuming dairy products and meat from infected animals.

The sub-populations of Susceptible animal (S_A), Exposed animal (E_A), and Infectious animal (I_A) make up the overall animal population, denoted by $\Omega_A(t)$.

The total Animal population becomes:

$\Omega_A(t) = S_A(t) + E_A(t) + I_A(t)$.

The total human population also represented by Ω_H, is divided into sub-populations of Susceptible humans (S_H), Vaccinated humans V_H, Exposed humans E_H, and Infected humans I_H.

The total human population is given by:

$\Omega_H(t) = S_H(t) + V_H(t) + E_H(t) + I_H(t)$.

Our current model is formulated by modifying the bovine tuberculosis model for human and animal which was developed by [5] which have seven compartments.

2.1. Model formulation. The list of variables and parameters used are as below

Symbol	Definition
S_H	Susceptible human population
E_H	Exposed human population
I_H	Infected human population
V_H	Vaccinated human population
S_A	Susceptible aniamal population
E_A	Exposed aniamal population
I_A	Infected aniamal population
C_e	Contamineted environment

TABLE 1. Model Variables and their defintions for bovine TB

Humans who are susceptible to bovine tuberculosis are recruited through birth and migration at a rate of Λ_H, and they contract the latent infection through contact with infected humans and

animals as well as through consumption of raw meat and dairy products from infected animals at a rate of Λ_H.

$$\lambda_H = \frac{\eta_1 I_H + \eta_2 I_A + \eta_3 C_e}{\Omega_H} \tag{1}$$

A portion of people obtain effective immunizations at a rate of κ, with $\kappa \in [0,1]$. The following latent infection of susceptible humans S_H is enhanced at a rate of λ_H by the exposed compartment E_H and decreased at a rate of γ_H by the advancement to the infectious stage. Due to disease-related deaths, human infections I_H grow at γ_H and diminish at α_H.

Every human compartment is subject to natural death at a rate of μ_H. Humans that have received vaccinations may transition to the exposed class at a pace of $d\lambda_H$ due to the vaccine's effectiveness's decreasing impact with $(1-d) \in [0,1]$. Humans may become susceptible and lose their immunity at a rate of ϕ.

At a rate of Λ_A, susceptible animals S_A are bred and migrated into populations, where they are latently infected with bovine tuberculosis through contact with diseased people and animals as well as dairy consumption.

$$\lambda_A = \frac{\eta_4 I_H + \eta_5 I_A + \eta_6 C_e}{\Omega_A} \tag{2}$$

After susceptible animals, exposed animals E_A increase at a rate of λ_A as S_A become latently infected. However, as they progress to the infectious stage, they begin to diminish at a rate of γ_A. Due to disease-induced death, infected animals I_A grow at a rate of γ_A and drop at a rate of α_A.

Natural mortality occurs at a rate of μ_A in every animal compartment. As sensitive humans and animals consume dairy products at rates of η_3 and η_6, respectively, infected animals produce dairy products or raw meat at rate of ρ and leak them out at rate of ω.

We will consider the fractional model using Caputo derivatives of order α such that $0 < \alpha < 1$.

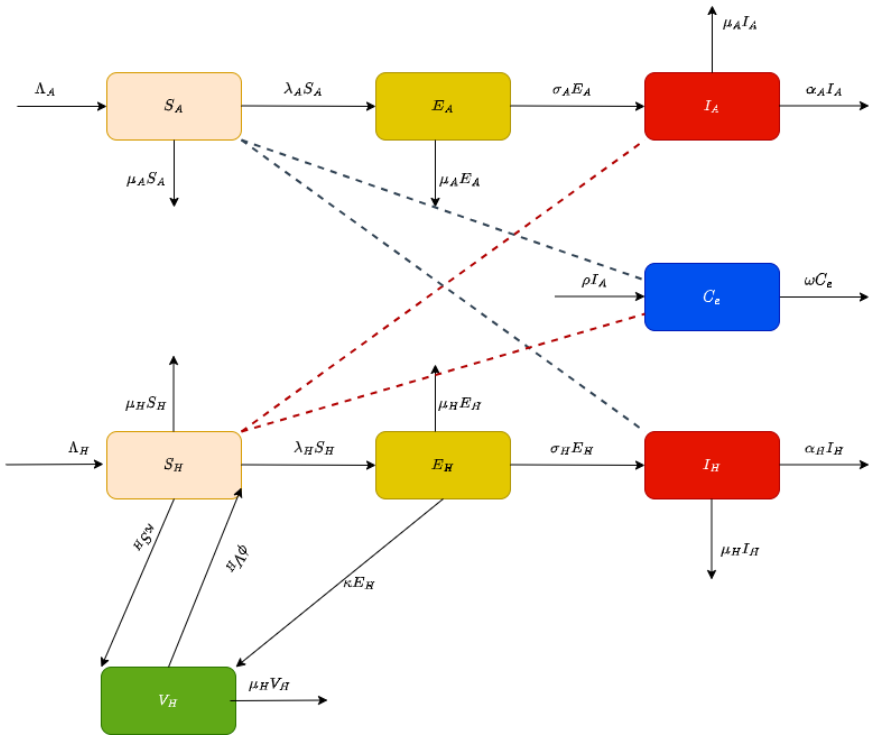


FIGURE 1. Schematic diagrams for bovine TB transmission among humans and animals

In our present work we will use Diethelm's approach [15], from Figure 1, we have the following system of fractional order equations:

(3)

$$
\begin{aligned}
{}_0^C D_t^\alpha S_H(t) &= (1-\kappa^\alpha)\Lambda_H^\alpha + \phi^\alpha V_H - \left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) S_H - \mu_H^\alpha S_H,\\
{}_0^C D_t^\alpha E_H(t) &= \left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) S_H + d\left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) V_H - (\mu_H^\alpha + \sigma_H^\alpha) E_H - \kappa^\alpha E_H,\\
{}_0^C D_t^\alpha V_H(t) &= \kappa^\alpha \Lambda_H^\alpha + \kappa^\alpha E_H - (\mu_H^\alpha + \phi^\alpha) V_H - d\left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) V_H,\\
{}_0^C D_t^\alpha I_H(t) &= \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha) I_H,\\
{}_0^C D_t^\alpha S_A(t) &= \Lambda_A^\alpha - \left(\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A}\right) S_A - \mu_A^\alpha S_A,\\
{}_0^C D_t^\alpha E_A(t) &= \left(\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A}\right) S_A - (\mu_A^\alpha + \sigma_A^\alpha) E_A,\\
{}_0^C D_t^\alpha I_A(t) &= \sigma_A^\alpha E_A - (\mu_A^\alpha + \gamma_A^\alpha) I_A,\\
{}_0^C D_t^\alpha C_e(t) &= \rho^\alpha I_A - \omega^\alpha C_e
\end{aligned}
$$

with initial condition,

$S_H(0) \geq 0, \quad E_H(0) \geq 0, \quad I_H(0) \geq 0, \quad V_H(0) \geq 0, \quad S_A(0) \geq 0, \quad E_A(0) \geq 0,$

$I_A(0) \geq 0, \quad C_e(0) \geq 0$

where ${}_0^C D^\alpha$ is the Caputo fractional derivative.

Note that, for simplification, in the following, we will use the notation D^α instead of ${}_0^C D^\alpha$.

3. Analysis of the Model

We look for invariant regions and evaluate the positivity of solutions to see if the model makes sense mathematically and epidemiologically. When the model's solutions are both positive and bounded, it becomes mathematically and biologically significant.

3.1. Invariant Region. The model solutions' viability is demonstrated by the invariant area. We use the initials Ω_H and Ω_A to represent the human and animal groups of individuals, respectively, to examine the viability of the model solutions.

Theorem 3.1. *Let* $\Psi = \{(S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t)) \in \mathbb{R}_+^8 :$
$0 \leq N_H \leq \frac{\Lambda_H}{\mu_H} \cup 0 \leq N_A \leq \frac{\Lambda_A}{\mu_A} \cup 0 \leq C_e \leq \frac{\Lambda_A}{\mu_A}\frac{\rho}{\omega}\}$
The feasible solution set $\{(S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t))\}$ *of the system equation of the model enter and bounded in the region* Ψ

Proof of Theorem 3.1. To prove this, let us consider the human population, animal population, and contaminatted environment separately.

• The fractional derivative of the total human population, obtained by adding all the human equations of the model (3), is given by

$$N_H(t) = S_H(t) + E_H(t) + V_H(t) + I_H(t)$$

$$D^\alpha N_H = D^\alpha S_H + D^\alpha E_H + D^\alpha V_H + D^\alpha I_H$$

$$D^\alpha N_H = \Lambda_H^\alpha + \phi^\alpha V_H - \lambda_H^\alpha S_H - \kappa^\alpha S_H - \mu_H^\alpha S_H + \lambda_H^\alpha S_H - (\mu_H^\alpha + \sigma_H^\alpha) E_H$$
$$+ \kappa^\alpha (S_H + E_H) - \mu_H^\alpha V_H + \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha) I_H$$

$$D^\alpha N_H = \Lambda_H^\alpha - \mu_H^\alpha S_H - \mu_H^\alpha E_H - \mu_H^\alpha V_H - \mu_H^\alpha I_H - \gamma_H^\alpha V_H - \gamma_H^\alpha I_H$$

$$D^{\alpha}N_H = \Lambda_H^{\alpha} - \mu_H^{\alpha}N_H - \gamma_H^{\alpha}I_H \tag{4}$$

$$D^{\alpha}N_H \leq \Lambda_H^{\alpha} - \mu_H^{\alpha}N_H \tag{5}$$

Note: To make simple the expressions, we'll do the calculations without α on the right hand side.

Let us take the Laplace transform [16] of equation (4) on both sides:

$$\mathscr{L}\left\{D_t^{\alpha}N_H(t)\right\}(s) + \mathscr{L}\left\{\mu_H N_H(t)\right\}(s) \geq \mathscr{L}\left\{\Lambda_H\right\}(s) \tag{6}$$

On the LHS:

$\mathscr{L}\left\{{}_aD_t^{\alpha}N_H(t)\right\}(s) = s^{\alpha}\mathscr{N}_H(t) - \sum_{k=0}^{n-1} s^{\alpha-k-1}N_H^{(k)}(0), \quad n-1 < \alpha \leq n$

Here $0 < \alpha < 1, \quad$ so $\quad n = 1,$

then, $\quad \mathscr{L}\left\{D_t^{\alpha}N_H(t)\right\}(s) = s^{\alpha}\mathscr{N}_H(s) - s^{\alpha-1}N_H(0)$, and

$$\mathscr{L}\left\{\mu_H N_H(t)\right\}(s) = \mu_H \mathscr{N}_H(s)$$

On the RHS

$$\begin{aligned}\mathscr{L}\left\{\Lambda_H\right\}(s) &= \Lambda_H \mathscr{L}\{1\} \\ &= \frac{\Lambda_H}{S}\end{aligned}$$

Now the equation (6) becomes:

$$\mathscr{L}\left\{D_t^{\alpha}N_H(t)\right\}(s) + \mathscr{L}\left\{\mu_H N_H(t)\right\}(s) \geq \mathscr{L}\left\{\Lambda_H\right\}(s) \tag{7}$$

$$s^{\alpha}\mathscr{N}_H(s) - s^{\alpha-1}N_H(0) + \mu_H \mathscr{N}_H(s) \geq \frac{\Lambda_H}{S} \tag{8}$$

$$\mathscr{N}_H(s)(s^{\alpha} + \mu_H) \geq \frac{\Lambda_H}{S} + s^{\alpha-1}N_H(0) \tag{9}$$

(10)

Hence, take $s^{\alpha-1}N_H(0) = 0$ at $t = 0$, [17]

then

$$\mathscr{N}_H(s) \geq \Lambda_H \frac{s^{-1}}{s^\alpha + \mu_H} \tag{11}$$

Taking the inverse Laplace transform of $\mathscr{N}_H(s)$, and by using the Mittag-Leffler function, we have:

$$\begin{aligned} N_H(t) &\leq \Lambda_H \mathscr{L}^{-1}\left\{\frac{s^{-1}}{s^\alpha + \mu_H}\right\} \\ &\leq \Lambda_H t^\alpha E_{\alpha,\alpha+1}(-\mu_H t^\alpha) \\ &\leq \frac{\Lambda_H}{\mu_H}\left[1 - E_\alpha(-\mu_H t^\alpha)\right] \end{aligned}$$

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H}\left[1 - E_\alpha(-\mu_H t^\alpha)\right] \tag{12}$$

We have $\mu_H > 0$ and then, as $t \longrightarrow 0$, thus $N_H(t) \longrightarrow \frac{\Lambda_H}{\mu_H} \geq 0$. Therefore

$$0 \leq N_H(t) \leq \frac{\Lambda_H}{\mu_H} \tag{13}$$

$$\Psi_H = \left\{(S_H, E_H, V_H, I_H) \in \mathbb{R}^4_+ : S_H + E_H + V_H + I_H \leq \frac{\Lambda_H^\alpha}{\mu_H^\alpha}\right\}. \tag{14}$$

• By the same approach, for animal population, we'll get:

$$\Psi_A = \left\{(S_A, E_A, I_A) \in \mathbb{R}^3_+ : S_H + E_H + I_H \leq \frac{\Lambda_A^\alpha}{\mu_A^\alpha}\right\}. \tag{15}$$

• For the case of contaminated environment:

$$D_t^\alpha C_e(t) = \rho^\alpha I_A - \omega^\alpha C_e, \tag{16}$$

with the assumption that $0 < I_A \leq \frac{\Lambda_A^\alpha}{\mu_A^\alpha}$.

Then we have from the equation (16)

$$D^\alpha C_e(t) \leq \rho^\alpha \frac{\Lambda_A^\alpha}{\mu_A^\alpha} - \omega^\alpha C_e \tag{17}$$

Now by taking the Laplace transform of the equation (17) on both sides and using the equality case, we have:

$$\mathscr{L}\left\{D_t^{\alpha} C_e(t)\right\}(s) \leq \mathscr{L}\left\{(\rho^{\alpha}\frac{\Lambda_A^{\alpha}}{\mu_A^{\alpha}}) - \omega^{\alpha} C_e(t)\right\}(s) \tag{18}$$

Following the same calculus approach in the human population case,

On the LHS,

$$\mathscr{L}\left\{D_t^{\alpha} C_e(t)\right\}(s) = s^{\alpha}\mathscr{C}_e(s) - s^{\alpha-1}C_e(0),$$

On the RHS,

$$\begin{aligned}\mathscr{L}\left\{(\rho\frac{\Lambda_A}{\mu_A}) - \omega C_e(t)\right\}(s) &= (\rho\frac{\Lambda_A}{\mu_A})\mathscr{L}\{1\} - \omega\mathscr{L}\{C_e(t)\} \\ &= \frac{(\rho\frac{\Lambda_A}{\mu_A})}{S} - \omega\mathscr{C}_e(s)\end{aligned}$$

Now the equation (18) becomes:

$$\mathscr{C}_e(s) = (\rho\frac{\Lambda_A}{\mu_A})\frac{s^{-1}}{s^{\alpha}+\omega} + \frac{s^{\alpha-1}}{s^{\alpha}+\omega}C_e(0) \tag{19}$$

Hence, take $s^{\alpha-1}C_e(0) = 0$ at $t = 0$,

then

$$\mathscr{C}_e(s) = (\rho\frac{\Lambda_A}{\mu_A})\frac{s^{-1}}{s^{\alpha}+\omega} \tag{20}$$

Taking the inverse Laplace transform of (20), we have:

$$\begin{aligned}C_e(t) &= (\rho\frac{\Lambda_A}{\mu_A})\mathscr{L}^{-1}\left\{\frac{s^{-1}}{s^{\alpha}+\omega}\right\} \\ &= (\rho\frac{\Lambda_A}{\mu_A})t^{\alpha}E_{\alpha,\alpha+1}(-\omega t^{\alpha})\end{aligned}$$

$$C_e(t) \leq \frac{\Lambda_A}{\mu_A}\frac{\rho}{\omega}\left[1 - E_{\alpha}(-\omega t^{\alpha})\right] \tag{21}$$

We have $\omega > 0$ and then, as $t \longrightarrow 0$, thus $C_e(t) \longrightarrow \frac{\Lambda_A}{\mu_A}\frac{\rho}{\omega} \geq 0$. Therefore

$$0 \leq C_e(t) \leq \frac{\Lambda_A}{\mu_A}\frac{\rho}{\omega} \tag{22}$$

and so

$$\Psi_{C_e} = \left\{C_e \in \mathbb{R}_+ : C_e \leq \frac{\Lambda_A^{\alpha}}{\mu_A^{\alpha}}\frac{\rho^{\alpha}}{\omega^{\alpha}}\right\} \tag{23}$$

The feasible region for the system of fractional differential equations in (3) is given by:

$$\Psi = \Psi_H \times \Psi_A \times \Psi_{c_e} \subset \mathbb{R}^4_+ \times \mathbb{R}^3_+ \times \mathbb{R}_+, \tag{24}$$

which is a positive invariant set.

This shows the boundedness of the solution of the model. □

3.2. Positivity of the Solution. In this section, we showed all the solution of the models Equation (3) remains positive for future time if their respective initial values are positive.

To establish this second result, we introduce the following lemma.

Lemma 3.1. *(Generalized Mean Value Theorem)* [18]

Suppose that $z(t) \in C[a,b]$ *and* ${}^C_0D^\alpha_t z(t) \in C[a,b]$ *for* $0 < \alpha \leq 1$, *then*

$$z(t) = z(a) + \frac{1}{\Gamma(\alpha)} {}^C_0D^\alpha_t z(\eta).(t-a)^\alpha, \tag{25}$$

where $a \leq \eta \leq t, \forall t \in (a,b]$.

Remark 3.1. *Assume that* $z(t) \in C[a,b]$ *and* ${}^C_0D^\alpha_t z(t) \in C[a,b]$ *for* $0 < \alpha \leq 1$. *It follows from Lemma* (3.1) *that if* ${}^C_0D^\alpha_t z(t) \geq 0, \forall t \in (a,b)$, *then* $z(t)$ *is increasing for* $\forall t \in [a,b]$, *and if* ${}^C_0D^\alpha_t z(t) \leq 0, \forall t \in (a,b)$ *then* $z(t)$ *is decreasing for* $\forall t \in [a,b]$

Theorem 3.2. *If* $S_H(0), E_H(0), V_H(0), I_H(0), S_A(0), E_A(0), I_A(0), C_e(0)$ *are positives, then* $S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t)$ *are also positives for all time* $t > 0$*;*

Proof of theorem 3.2. Let us take all the equations of the model in Equation (3) at $t = 0$, we have:

$$ {}^C_0D^\alpha_t S_H|_{S_H=0} = (1-\kappa^\alpha)\Lambda^\alpha_H + \phi^\alpha V_H \geq 0 \tag{26}$$

$$ {}^C_0D^\alpha_t E_H|_{E_H=0} = \lambda_H S_H + d\lambda_H V_H \geq 0 \tag{27}$$

$$ {}^C_0D^\alpha_t V_H|_{V_H=0} = \kappa^\alpha \Lambda^\alpha_H + \kappa^\alpha E_H \geq 0 \tag{28}$$

$$ {}^C_0D^\alpha_t I_H|_{I_H=0} = \sigma^\alpha_H E_H \geq 0 \tag{29}$$

$$ {}^C_0D^\alpha_t S_A|_{S_A=0} = \Lambda^\alpha_A > 0 \tag{30}$$

$$ {}^C_0D^\alpha_t E_A|_{E_A=0} = \lambda_A S_A \geq 0 \tag{31}$$

$$ {}_0^C D_t^\alpha I_A|_{I_A=0} = \sigma_A^\alpha E_A \geq 0 \tag{32} $$

$$ {}_0^C D_t^\alpha C_e|_{C_e=0} = \rho^\alpha I_A \geq 0 \tag{33} $$

Since $S_H(0), E_H(0), V_H(0), I_H(0), S_A(0), E_A(0), I_A(0), C_e(0)$ are positives, according to (26)-(33) and the remark (3.1), the solution $(S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t))$ can't scape from the hyperplanes of $S_H = 0, E_H = 0, V_H = 0, V_H = 0, I_H = 0, S_A = 0$
$E_A = 0, I_A = 0$, and $C_e = 0$. Therefore, all the solutions of the model with initial conditions in Ψ remain in Ψ for all $t > 0$. Thus, this region is a positive invariant set.

The model (3) is mathematically and epidemiologically meaningful; therefore, we can consider the flow generated by the model for analysis. □

3.3. Disease-Free Equilibrium (DFE), for the model of bTB. The situation in which there are no diseases affecting the populace is known as the disease-free equilibrium point. According to Φ_0, the disease-free equilibrium is established when bTB is absent from both the human and animal populations.

$$ \begin{cases} {}_0^C D_t^\alpha S_H(t) &= 0, \\ {}_0^C D_t^\alpha E_H(t) &= 0, \\ {}_0^C D_t^\alpha V_H(t) &= 0, \\ {}_0^C D_t^\alpha I_H(t) &= 0, \\ {}_0^C D_t^\alpha S_A(t) &= 0, \\ {}_0^C D_t^\alpha E_A(t) &= 0, \\ {}_0^C D_t^\alpha I_A(t) &= 0, \\ {}_0^C D_t^\alpha C_e(t) &= 0 \end{cases} \tag{34} $$

After some calculus, we get:

$$ \Phi_0 = \left(\frac{\Lambda_H^\alpha (\phi^\alpha + (1-\kappa^\alpha)\mu_H^\alpha)}{\mu_H(\mu_H^\alpha + \phi^\alpha)}, 0, \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}, 0, \frac{\Lambda_A^\alpha}{\mu_A^\alpha}, 0, 0, 0 \right) \tag{35} $$

3.4. The Basic Reproduction Number. The basic reproduction number R_0 describes the typical number of new cases that a single infectious person creates when they are introduced into a community that is completely susceptible [19, 20, 21]. It establishes if the illness spreads or

disappears in the community. When the fundamental reproduction number R_0 is less than 1, the disease disappears from the population. If R_0 is more than 1, the disease continues. This is true because the disease survives when an infectious person is brought to a community that is completely vulnerable to infection[22, 23].

To determine the basic reproduction number R_0 , we use the next-generation matrix technique while accounting for new infections and transfer terms.[19, 22, 24]. The R_0 is expressed as the greatest eigenvalue if the new infectious and transfer terms for bTB are indicated by F_i and V_i, respectively. We have,

$$R_0 = \rho(FV^{-1})$$

where

$$F = \left| \frac{\partial F_i x(0)}{\partial x_j} \right|, \quad V = \left| \frac{\partial V_i x(0)}{\partial x_j} \right|,$$

ρ denotes here the spectral radius of a matrix which is the greatest eigenvalue of a given matrix.

We only take into account the infectious, the exposed, and contaminated environnement classes in the system of fractional differential equations in (3) using the Next-Generation Matrix.

$$\begin{cases} {}_0^C D_t^\alpha E_H(t) &= \lambda_H^\alpha S_H + d\lambda_H^\alpha V_H - (\mu_H^\alpha + \sigma_H^\alpha)E_H - \kappa^\alpha E_H, \\ {}_0^C D_t^\alpha I_H(t) &= \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha)I_H, \\ {}_0^C D_t^\alpha E_A(t) &= \lambda_A^\alpha S_A - (\mu_A^\alpha + \sigma_A^\alpha)E_A, \\ {}_0^C D_t^\alpha I_A(t) &= \sigma_A^\alpha E_A - (\mu_A^\alpha + \gamma_A^\alpha)I_A, \\ {}_0^C D_t^\alpha C_e(t) &= \rho^\alpha I_A - \omega^\alpha C_e \end{cases} \tag{36}$$

Let F_i represent the number of new infections entering the system and V_i represent the number of infections leaving the system as a result of births or deaths.

$$F_i = \begin{bmatrix} (\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H})S_H + d(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H})V_H \\ 0 \\ (\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A})S_A \\ 0 \\ 0 \end{bmatrix}$$

$$V_i = \begin{bmatrix} (\mu_H^\alpha + \sigma_H^\alpha)E_H + \kappa^\alpha E_H \\ -\sigma_H^\alpha E_H + (\mu_H^\alpha + \gamma_H^\alpha)I_H \\ (\mu_A^\alpha + \sigma_A^\alpha)E_A \\ -\sigma_A^\alpha E_A + (\mu_A^\alpha + \gamma_A^\alpha)I_A \\ -\rho^\alpha I_A + \omega^\alpha C_e \end{bmatrix}$$

Now let'us express the jacobien matrix of F_i and V_i by F and V respectively.

$$F = \begin{bmatrix} 0 & \frac{\eta_1 S_H}{\Omega_H} + d\frac{\eta_1 V_H}{\Omega_H} & 0 & \frac{\eta_2 S_H}{\Omega_H} + d\frac{\eta_2 V_H}{\Omega_H} & \frac{\eta_3 S_H}{\Omega_H} + d\frac{\eta_3 V_H}{\Omega_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\eta_4 S_A}{\Omega_A} & 0 & \frac{\eta_5 S_A}{\Omega_A} & \frac{\eta_6 S_A}{\Omega_A} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} (\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) & 0 & 0 & 0 & 0 \\ -\sigma_H^\alpha & (\mu_H^\alpha + \gamma_H^\alpha) & 0 & 0 & 0 \\ 0 & 0 & (\mu_A^\alpha + \sigma_A^\alpha) & 0 & 0 \\ 0 & 0 & -\sigma_A^\alpha & (\mu_A^\alpha + \gamma_A^\alpha) & 0 \\ 0 & 0 & 0 & -\rho^\alpha & \omega^\alpha \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)} & 0 & 0 & 0 & 0 \\ \frac{\sigma_H^\alpha}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)} & \frac{1}{(\mu_H^\alpha+\gamma_H^\alpha)} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\mu_A^\alpha+\sigma_A^\alpha)} & 0 & 0 \\ 0 & 0 & \frac{\sigma_A^\alpha}{(\mu_A^\alpha+\sigma_A^\alpha)(\mu_A^\alpha+\gamma_A^\alpha)} & \frac{1}{(\mu_A^\alpha+\gamma_A^\alpha)} & 0 \\ 0 & 0 & \frac{\rho^\alpha\sigma_A^\alpha}{(\mu_A^\alpha+\sigma_A^\alpha)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha} & \frac{\rho^\alpha}{(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha} & \frac{1}{\omega^\alpha} \end{bmatrix}$$

Note: To simplify the claculus we'll make the folowing notations and leave α, the order of derivative.

$$R_0 = FV^{-1} = \begin{bmatrix} A_1 & A_2 & A_3 & A_4 & A_5 \\ 0 & 0 & 0 & 0 & 0 \\ B_1 & B_2 & B_3 & B_4 & B_5 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{37}$$

where,

$$A_1 = \frac{\eta_1\sigma_H(dV_H + S_H)}{(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)}$$
$$A_2 = \frac{dV_H\eta_1 + \eta_1 S_H}{\mu_H + \gamma_H}$$
$$A_3 = \frac{(dV_H\eta_2 + \eta_2 S_H)\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)} + \frac{(dV_H\eta_3 + \eta_3 S_H)\rho\,\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)\omega}$$
$$A_4 = \frac{dV_H\eta_2 + \eta_2 S_H}{\mu_A + \gamma_A} + \frac{(dV_H\eta_3 + \eta_3 S_H)\rho}{(\mu_A + \gamma_A)\omega}$$
$$A_5 = \frac{d\eta_3 V_H + \eta_3 S_H}{\omega}$$

$$B_1 = \frac{\eta_4\sigma_H S_A}{(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)}$$
$$B_2 = \frac{\eta_4 S_A}{\mu_H + \gamma_H}$$
$$B_3 = \frac{\eta_5 S_A\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)} + \frac{\eta_6 S_A\rho\,\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)\omega}$$
$$B_4 = \frac{\eta_5 S_A}{\mu_A + \gamma_A} + \frac{\eta_6 S_A\rho}{(\mu_A + \gamma_A)\omega}$$
$$B_5 = \frac{\eta_6 S_A}{\omega}$$

Now let us compute the eigenvalues of FV^{-1} and selecte the dominant eigenvalue.

Let X represent the eigenvalue of the matrix

$$\begin{vmatrix} A_1 - X & A_2 & A_3 & A_4 & A_5 \\ 0 & -X & 0 & 0 & 0 \\ B_1 & B_2 & B_3 - X & B_4 & B_5 \\ 0 & 0 & 0 & -X & 0 \\ 0 & 0 & 0 & 0 & -X \end{vmatrix} = 0 \tag{38}$$

The equation (38) is equivalent to:

$$X^3 \begin{vmatrix} A_1 - X & A_3 \\ B_1 & B_3 - X \end{vmatrix} = 0 \tag{39}$$

We have the following characteristic equation:

$$X^3[X^2 - (A_1 + B_3)X + A_1B_3 - A_3B_1] = 0 \tag{40}$$

The maximum eigenvalue is then:

$$X = \frac{A_1 + B_3}{2} + \sqrt{\frac{(A_1 + B_3)^2 - 4(A_1 B_3 - A_3 B_1)}{4}}$$
$$X = \frac{A_1 + B_3}{2} + \frac{\sqrt{(A_1 - B_3)^2 + 4A_3 B_1}}{2} \tag{41}$$

Now let us evaluate A_1, A_3, B_1 and B_3 at the DFE Φ_0:

$$A_1 = \frac{\eta_1 \sigma_H \left(d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H\right)}{\left(\mu_H + \phi\right)\left(\mu_H + \sigma_H + \kappa\right)\left(\mu_H + \gamma_H\right)}$$

$$A_3 = \frac{\sigma_A \left(d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H\right)\left(\omega \eta_2 + \rho \eta_3\right)}{\left(\mu_H + \phi\right)\left(\mu_A + \sigma_A\right)\left(\mu_A + \gamma_A\right)\omega}$$

$$B_1 = \frac{\eta_4 \sigma_H}{\left(\mu_H + \sigma_H + \kappa\right)\left(\mu_H + \gamma_H\right)}$$

$$B_3 = \frac{\sigma_A(\eta_5 \omega + \eta_6 \rho)}{\left(\mu_A + \sigma_A\right)\left(\mu_A + \gamma_A\right)\omega}$$

By substituting A_1, A_3, B_1 and the B_3, we have:

$$R_1 = A_1 + B_3 = \frac{\eta_1 \sigma_H \left(d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H\right)}{\left(\mu_H + \phi\right)\left(\mu_H + \sigma_H + \kappa\right)\left(\mu_H + \gamma_H\right)} + \frac{\sigma_A(\eta_5 \omega + \eta_6 \rho)}{\left(\mu_A + \sigma_A\right)\left(\mu_A + \gamma_A\right)\omega} \tag{42}$$

$$R_2 = A_1 - B_3 = \frac{\eta_1 \sigma_H \left(d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H\right)}{\left(\mu_H + \phi\right)\left(\mu_H + \sigma_H + \kappa\right)\left(\mu_H + \gamma_H\right)} - \frac{\sigma_A(\eta_5 \omega + \eta_6 \rho)}{\left(\mu_A + \sigma_A\right)\left(\mu_A + \gamma_A\right)\omega} \tag{43}$$

$$R_3 = A_3 B_1 = \frac{\eta_4 \sigma_H \sigma_A \left(d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H\right)\left(\omega \eta_2 + \rho \eta_3\right)}{\omega \left(\mu_H + \phi\right)\left(\mu_A + \sigma_A\right)\left(\mu_A + \gamma_A\right)\left(\mu_H + \sigma_H + \kappa\right)\left(\mu_H + \gamma_H\right)} \tag{44}$$

$$R_0 = \frac{R_1}{2} + \frac{\sqrt{R_2^2 + 4R_3}}{2} \tag{45}$$

In equation (42), the terms $\frac{1}{(\mu_H + \sigma_H + \kappa)}$ and $\frac{1}{(\mu_A + \sigma_A)}$ stand for the average amount of time each human and animal spend in their respective exposed classes, $\frac{1}{(\mu_H + \phi)}$, the average amount of time each human spend in the vaccineted class, $\frac{1}{(\mu_H + \gamma_H)}$ and $\frac{1}{(\mu_A + \gamma_A)}$ for the average amount of time

each infectious human and animal spend in their infectious classes, $\frac{\eta_1\sigma_H[\phi+\mu_H(1+d\kappa-\kappa)]}{(\mu_H+\phi)(\mu_H+\gamma_H)(\mu_H+\sigma_H+\kappa)}$ is the percentage of infected humans who develop bTB and move from the exposed class to the infectious class after coming into contact with infectious humans and animals, respectively, and $\frac{\sigma_A(\omega\eta_5+\rho\eta_6)}{\omega(\mu_A+\gamma_A)(\mu_A+\sigma_A)}$ represents the overall proportion of diseased animals that pass from the exposed class to the infectious class as a result of interaction with infected animals and consumption of infectious dairy products.

The total of the proportions of infected people who contract bTB through contact with diseased animals and after ingesting infectious meat or dairy products is given by (43) :

$$\frac{\eta_4\sigma_H\sigma_A\left(d\kappa\mu_H-\kappa\mu_H+\phi+\mu_H\right)\left(\omega\eta_2+\rho\eta_3\right)}{\omega\left(\mu_H+\phi\right)\left(\mu_A+\sigma_A\right)\left(\mu_A+\gamma_A\right)\left(\mu_H+\sigma_H+\kappa\right)\left(\mu_H+\gamma_H\right)}.$$

3.5. Local stability Analysis for Disease-Free Equilibrium (DFE). To assess the local stability of a disease-free equilibrium when trace and determinant are used, we apply the linearization method like in [5]. If the eigenvalues of the Jacobien matrix are negative or have a negative real part, disease-free equilibrium is considered to be locally asymptotically stable.

Theorem 3.3. *If all of the eigenvalues of the $J(\Phi_0)$ satisfy the requirement that $|arg\lambda_j| > \frac{\alpha\pi}{2}$, where $j = 1,2,3\cdots$, and $0 < \alpha \leq 1$. Then Φ_0 is locally asymptotically stable.*

Proof of theorem 3.3. Taking the partial derivatives of each equation with respect to each variable, we get:

$$J(\mathbf{x}) = \begin{bmatrix} -\lambda_H^\alpha-\mu_H^\alpha & 0 & \phi^\alpha & -\frac{\eta_1^\alpha S_H}{\Omega_H} & 0 & 0 & -\frac{\eta_2^\alpha S_H}{\Omega_H} & -\frac{\eta_3^\alpha S_H}{\Omega_H} \\ \lambda_H^\alpha & -\mu_H^\alpha-\sigma_H^\alpha-\kappa^\alpha & d\lambda_H^\alpha & d\frac{\eta_1^\alpha S_H}{\Omega_H} & 0 & 0 & d\frac{\eta_2^\alpha S_H}{\Omega_H} & d\frac{\eta_3^\alpha S_H}{\Omega_H} \\ 0 & \kappa^\alpha & -\mu_H^\alpha-\phi^\alpha-d\lambda_H^\alpha & -d\frac{\eta_1^\alpha V_H}{\Omega_H} & 0 & 0 & -d\frac{\eta_2^\alpha V_H}{\Omega_H} & -d\frac{\eta_3^\alpha V_H}{\Omega_H} \\ 0 & \sigma_H^\alpha & 0 & -(\mu_H^\alpha+\gamma_H^\alpha) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\eta_4^\alpha S_A}{N_A} & -\lambda_1^\alpha-\mu_A^\alpha & 0 & -\frac{\eta_5^\alpha S_A}{\Omega_A} & -\frac{\eta_6^\alpha S_A}{\Omega_A} \\ 0 & 0 & 0 & \frac{\eta_4^\alpha S_A}{\Omega_A} & \lambda_A^\alpha & -\mu_A-\sigma_A & \frac{\eta_5^\alpha S_A}{\Omega_A} & \frac{\eta_6^\alpha S_A}{\Omega_A} \\ 0 & 0 & 0 & 0 & 0 & \sigma_A^\alpha & -\mu_A^\alpha-\gamma_A^\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix} \tag{46}$$

where $\mathbf{x} = [S_H, E_H, V_H, I_H, S_A, E_A, I_A, C_e]$ is the vector of variables, and $J(\mathbf{x})_{ij}$ represents the partial derivative of the i-th equation with respect to the j-th variable.

After the Jacobian has been evaluated at DFE Φ_0, we have

(47)
$$J(\Phi_0) = \begin{bmatrix} -\mu_H^\alpha & 0 & \phi^\alpha & \eta_1^\alpha & 0 & 0 & -\eta_2^\alpha & -\eta_3^\alpha \\ 0 & -\mu_H^\alpha - \sigma_H^\alpha - \kappa^\alpha & 0 & d\eta_1^\alpha & 0 & 0 & d\eta_2^\alpha & d\eta_3^\alpha \\ 0 & \kappa^\alpha & -\mu_H^\alpha - \phi^\alpha & -d\frac{\eta_1^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} & 0 & 0 & -d\frac{\eta_2^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} & -d\frac{\eta_3^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} \\ 0 & \sigma_H^\alpha & 0 & -\mu_H^\alpha - \gamma_H^\alpha & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\eta_4^\alpha & -\mu_A^\alpha & 0 & -\eta_5^\alpha & -\eta_6^\alpha \\ 0 & 0 & 0 & \eta_4^\alpha & 0 & -\mu_A - \sigma_A & \eta_5^\alpha & \eta_6^\alpha \\ 0 & 0 & 0 & 0 & 0 & \sigma_A^\alpha & -\mu_A^\alpha - \gamma_A^\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix}$$

The Matrix (47) has negatitive eigenvalues $-\mu_H^\alpha$, $-\mu_A^\alpha$ and $-\mu_H^\alpha - \phi^\alpha$, and those three eigenvalues satisfy the condition: $|arg\lambda_j| > \frac{\alpha\pi}{2}$ for all $0 < \alpha \leq 1$.

Matrix (47) reduces now to:

(48)
$$R = \begin{bmatrix} -\mu_H^\alpha - \sigma_H^\alpha - \kappa^\alpha & d\eta_1^\alpha & 0 & d\eta_2^\alpha & d\eta_3^\alpha \\ \sigma_H^\alpha & -\mu_H^\alpha - \gamma_H^\alpha & 0 & 0 & 0 \\ 0 & \eta_4^\alpha & -\mu_A - \sigma_A & \eta_5^\alpha & \eta_6^\alpha \\ 0 & 0 & \sigma_A^\alpha & -\mu_A^\alpha - \gamma_A^\alpha & 0 \\ 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix}$$

We employ trace tr and determinant det to examine matrix R. If the determinant is positive $\det(R) > 0$ and the trace is negative $tr(K) < 0$, then the disease-free equilibrium is locally stable. The trace of the matrix R is given by:

(49)
$$tr(R) = -\left((\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) + (\mu_H^\alpha + \gamma_H^\alpha) + (\mu_A + \sigma_A) + (\mu_A^\alpha + \gamma_A^\alpha) + \omega^\alpha\right) < 0$$

The determinant of R is given by:

(50)
$$\det(R) = -(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) \begin{vmatrix} -\mu_H^\alpha - \gamma_H^\alpha & 0 & 0 & 0 \\ \eta_4^\alpha & -\mu_A - \sigma_A & \eta_5^\alpha & \eta_6^\alpha \\ 0 & \sigma_A^\alpha & -\mu_A^\alpha - \gamma_A^\alpha & 0 \\ 0 & 0 & \rho^\alpha & -\omega^\alpha \end{vmatrix}$$

$$-\sigma_H^\alpha \begin{vmatrix} d\eta_1^\alpha & 0 & d\eta_2^\alpha & d\eta_3^\alpha \\ \eta_4^\alpha & -\mu_A-\sigma_A & \eta_5^\alpha & \eta_6^\alpha \\ 0 & \sigma_A^\alpha & -\mu_A^\alpha-\gamma_A^\alpha & 0 \\ 0 & 0 & \rho^\alpha & -\omega^\alpha \end{vmatrix}$$

$$= (\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)\left[(\sigma_A^\alpha(\omega^\alpha\eta_5^\alpha+\rho^\alpha\eta_6^\alpha)-(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha\right]$$

$$+\sigma_H^\alpha d\eta_1^\alpha(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha + d\sigma_H^\alpha\sigma_A^\alpha\left[\omega^\alpha(\eta_2^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_5^\alpha)+\rho^\alpha(\eta_3^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_6^\alpha)\right]$$

Let $\det(R)=0$ then,

(51)

$$\begin{aligned} 0 =& \sigma_A^\alpha(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)(\omega^\alpha\eta_5^\alpha+\rho^\alpha\eta_6^\alpha) \\ & -(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha+\sigma_H^\alpha d\eta_1^\alpha(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha \\ & +d\sigma_H^\alpha\sigma_A^\alpha\left[\omega^\alpha(\eta_2^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_5^\alpha)+\rho^\alpha(\eta_3^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_6^\alpha)\right] \\ =& \frac{\sigma_A^\alpha(\omega^\alpha\eta_5^\alpha+\rho^\alpha\eta_6^\alpha)}{(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)}+\frac{\sigma_H^\alpha d\eta_1^\alpha\omega^\alpha}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)} \\ & +\frac{d\sigma_H^\alpha\sigma_A^\alpha\left[\omega^\alpha(\eta_2^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_5^\alpha)+\rho^\alpha(\eta_3^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_6^\alpha)\right]}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha}-1 \end{aligned}$$

Thus $\det(R)>0$ if

$$\frac{\sigma_A^\alpha(\omega^\alpha\eta_5^\alpha+\rho^\alpha\eta_6^\alpha)}{(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)}+\frac{\sigma_H^\alpha d\eta_1^\alpha\omega^\alpha}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)}+\frac{d\sigma_H^\alpha\sigma_A^\alpha\left[\omega^\alpha(\beta_2^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_5^\alpha)+\rho^\alpha(\eta_3^\alpha\eta_4^\alpha-\eta_1^\alpha\eta_6^\alpha)\right]}{(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)(\mu_H^\alpha+\gamma_H^\alpha)(\mu_A+\sigma_A)(\mu_A^\alpha+\gamma_A^\alpha)\omega^\alpha}>1. \tag{52}$$

Then the conditions of trace and determinant are proved, thus the others eigenvalues have negative real part. So that: $|arg\lambda_j|>\frac{\alpha\pi}{2}$ for all $0<\alpha\leq 1$.

Conclusion: The disease-free equilibrium ψ_0 of the model (3) is locally asymptotically stable whenever the condition (52) holds as well as $R_0<1$ and it is unstable when $R_0>1$. □

3.6. Global Stability of the Disease-Free Equilibrium. The global asymptotically stability (GAS) of the disease-free state of the model is investigated using the theorem by [25, 26, 27]. So from the model (3) we have:

$$\begin{cases} \dfrac{dU}{dt} &= F(U,Z), \\ \\ \dfrac{dZ}{dt} &= G(U,Z), \quad \text{with} \quad G(U,0)=0. \end{cases} \tag{53}$$

where

- $U = (S_H, V_H, S_A)$ is the number of uninfected individuals, and
- $Z = (E_H, I_H, E_A, I_A, C_e)$ represents the number of infected individuals

Let U^* be the disease-free equilibrium (DFE) of the system $\dfrac{dU}{dt} = F(U,0)$, and

$$U^* = \left(\frac{\Lambda_H^\alpha (\phi^\alpha + (1-\kappa^\alpha)\mu_H^\alpha)}{\mu_H(\mu_H^\alpha + \phi^\alpha)}, \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}, \frac{\Lambda_A^\alpha}{\mu_A^\alpha} \right),$$

If $R_0 < 1$ (which is locally asymptotically stable (LAS)), and the following two assumptions A1 and A2 hold, the Disease-Free Equilibrium (DFE) point Φ_0 of the model is guaranteed to be GAS:

- A1: For $\dfrac{dU}{dt} = F(U,0)$, U^* is globally GAS for the model (3) provided that $R_0 < 1$ (LAS) and assumptions A1 and A2 hold.
- $A2 : G(U,Z) = AZ - G^*(U,Z), \quad G^*(U,Z) \geq 0, \quad \forall (U,Z) \in \Psi.$
  The region where the model makes biological sense is Ψ_0, and $A = \dfrac{\partial G(\Phi_0)}{\partial Z}$ is an M-matrix (the nondiagonal entries are nonnegative).

The following theorem is true if the model equation (3) satisfies the above two requirements.

Theorem 3.4. *The disease-free equilibrium point, Φ_0 is globally asymptotically stability (GAS) for the model* (3) *provided that $R_0 < 1$ locally asymptotically stable (LAS) and the conditions A1 and A2 hold.*

Proof of theorem 3.4. Let us show that the condition A1 and A2 hold when $R_0 < 1$, to do that, we need to show that $U \longrightarrow U^*$.

$$F(U,0) = \begin{cases} {}_0^C D_t^\alpha S_H(t) = \Lambda_H^\alpha + \phi^\alpha V_H - \kappa^\alpha \Lambda_H^\alpha - \mu_H^\alpha S_H, \\ {}_0^C D_t^\alpha V_H(t) = \kappa^\alpha \Lambda_H^\alpha - (\mu_H^\alpha + \phi^\alpha) V_H, \\ {}_0^C D_t^\alpha S_A(t) = \Lambda_A^\alpha - \mu_A^\alpha S_A \end{cases} \tag{54}$$

The second and the third equation of the equation (54) are the α's order linear ODE's and we have their solution like following:

${}_0^C D_t^\alpha S_H(t) = \Lambda_H^\alpha + \phi^\alpha V_H - \kappa^\alpha \Lambda_H^\alpha - \mu_H^\alpha S_H$, using the Laplace transform

$$\mathscr{L}\left\{D_t^\alpha S_A(t)\right\}(W) = \mathscr{L}\left\{\Lambda_A^\alpha - \mu_A^\alpha S_A(t)\right\}(W) \Longrightarrow$$

$$\mathscr{L}\left\{D_t^\alpha S_A(t)\right\}(W) + \mathscr{L}\left\{\mu_A^\alpha S_A(t)\right\}(W) = \mathscr{L}\left\{\Lambda_A^\alpha\right\} \Longrightarrow \tag{55}$$

$$W^\alpha S_A(W) - D^{-(1-\alpha)} S_A(0) + \mu_A^\alpha S_A(W) = \frac{\Lambda_A^\alpha}{W}$$

At $t = 0$ $D^{-(1-\alpha)} S_A(0) = 0$. Then

$$(W^\alpha + \mu_A^\alpha) S_A(W) = \frac{\Lambda_A^\alpha}{W} \Longrightarrow S_A(W) = \frac{\Lambda_A^\alpha}{W(W^\alpha + \mu_A^\alpha)}$$

Now by taking the Laplace inverse transform of of $S_A(W)$ and using the Mittag-Leffler function we obtain:

$$S_A(t) = \frac{\Lambda_A^\alpha}{\mu_A^\alpha}\left[1 - E_\alpha(-\mu_A^\alpha t^\alpha)\right] \quad \text{with} \quad \mu_A^\alpha > O$$

Then we have: $S_A(t) \longrightarrow \dfrac{\Lambda_A^\alpha}{\mu_A^\alpha}$ if $t \longrightarrow \infty$.

By the same method we obtain:

$$V_H(t) = \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}\left[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha) t^\alpha)\right] \quad \text{with} \quad (\mu_A^\alpha + \phi^\alpha) > O$$

Then we have: $V_H(t) \longrightarrow \dfrac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}$ if $t \longrightarrow \infty$.

Now by substuting $V_H(t)$ in the first equation of (54) yields:

$$D_t^\alpha S_H(t) = \Lambda_H^\alpha(1 - \kappa^\alpha) - \mu_H^\alpha S_H + \phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}\left[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha) t^\alpha)\right]. \tag{56}$$

Let us take the Laplace transform of (56):

(57)

$$\mathscr{L}\left\{D_t^\alpha S_H(t)\right\}(W) = \mathscr{L}\left\{\Lambda_H^\alpha(1 - \kappa^\alpha) - \mu_H^\alpha S_H + \phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}\left[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha) t^\alpha)\right]\right\}(W) \Longrightarrow$$

$$\mathscr{L}\left\{D_t^\alpha S_H(t)\right\}(W) + \mathscr{L}\left\{\mu_H^\alpha S_H(t)\right\}(W) = \mathscr{L}\left\{\Lambda_H^\alpha(1 - \kappa^\alpha)\right\} + \mathscr{L}\left\{\phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}\left[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha) t^\alpha)\right]\right\} \Longrightarrow$$

$$W^\alpha S_H(W) - D^{-(1-\alpha)} S_H(0) + \mu_H^\alpha S_H(W) = \frac{\Lambda_H^\alpha(1 - \kappa^\alpha)}{W} + \phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}\left[\frac{1}{W} - \frac{W^{\alpha-1}}{W^\alpha + (\mu_H^\alpha + \phi^\alpha)}\right]$$

At $t = 0$ $D^{-(1-\alpha)} S_H(0) = 0$.

Then

$$
\begin{aligned}
&(W^\alpha+\mu_H^\alpha)S_H(W)=\frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{W}+\phi^\alpha\frac{\kappa^\alpha\Lambda_H^\alpha}{\mu_H^\alpha+\phi^\alpha}\left[\frac{1}{W}-\frac{W^{\alpha-1}}{W^\alpha+(\mu_H^\alpha+\phi^\alpha)}\right]\Longrightarrow\\
&S_H(W)=\frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{W(W^\alpha+\mu_H^\alpha)}+\phi^\alpha\frac{\kappa^\alpha\Lambda_H^\alpha}{(\mu_H^\alpha+\phi^\alpha)(W^\alpha+\mu_H^\alpha)W}-\frac{\phi^\alpha\kappa^\alpha\Lambda_H^\alpha}{(\mu_H^\alpha+\phi^\alpha)}\frac{1}{W^\alpha+\mu_H^\alpha}\times\frac{W^{\alpha-1}}{W^\alpha+(\mu_H^\alpha+\phi^\alpha)}
\end{aligned}
\tag{58}
$$

Now by taking the Laplace Inverse Transform, we obtain

$$
\begin{aligned}
S_H(t)=&\frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{\mu_H^\alpha}\left[1-E_\alpha(-\mu_H^\alpha t^\alpha)\right]+\frac{\phi^\alpha\kappa^\alpha\Lambda_H^\alpha}{\mu_H^\alpha(\mu_H^\alpha+\phi^\alpha)}\left[1-E_\alpha(-\mu_H^\alpha t^\alpha)\right]\\
&-\frac{\phi^\alpha\kappa^\alpha\Lambda_H^\alpha}{(\mu_H^\alpha+\phi^\alpha)}\times t^{\alpha-1}E_{\alpha,\alpha}(-\mu_H^\alpha t^\alpha)\times E_{\alpha,1}\left[-(\mu_H^\alpha+\phi^\alpha)\right]
\end{aligned}
\tag{59}
$$

$$
\begin{aligned}
\lim_{t\to\infty}S_H(t)&=\frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{\mu_H^\alpha}+\frac{\phi^\alpha\kappa^\alpha\Lambda_H^\alpha}{\mu_H^\alpha(\mu_H^\alpha+\phi^\alpha)}\\
&=\frac{\Lambda_H^\alpha\left[\phi^\alpha+\mu_H^\alpha(1-\kappa^\alpha)\right]}{\mu_H^\alpha(\mu_H^\alpha+\phi^\alpha)}
\end{aligned}
\tag{60}
$$

Thus all points with respect to this conditions converge at

$$
U^*=\left(\frac{\Lambda_H^\alpha\left(\phi^\alpha+(1-\kappa^\alpha)\mu_H^\alpha\right)}{\mu_H(\mu_H^\alpha+\phi^\alpha)},\frac{\kappa^\alpha\Lambda_H^\alpha}{\mu_H^\alpha+\phi^\alpha},\frac{\Lambda_A^\alpha}{\mu_A^\alpha}\right)
$$

. Hence U^* is globally asymptotically stable.

For the next step, we have:

$$
G(U,Z)=\begin{cases}
G1(U,Z)=\left(\dfrac{\eta_1^\alpha I_H+\eta_2^\alpha I_A+\eta_3^\alpha C_e}{\Omega_H}\right)S_H+d\left(\dfrac{\eta_1^\alpha I_H+\eta_2^\alpha I_A+\eta_3^\alpha C_e}{\Omega_H}\right)V_H-(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha)E_H\\
G2(U,Z)=\sigma_H^\alpha E_H-(\mu_H^\alpha+\gamma_H^\alpha)I_H,\\
G3(U,Z)=\left(\dfrac{\eta_4^\alpha I_H+\eta_5^\alpha I_A+\eta_6^\alpha C_e}{\Omega_A}\right)S_A-(\mu_A^\alpha+\sigma_A^\alpha)E_A,\\
G4(U,Z)=\sigma_A^\alpha E_A-(\mu_A^\alpha+\gamma_A^\alpha)I_A,\\
G5(U,Z)=\rho^\alpha I_A-\omega^\alpha C_e
\end{cases}
\tag{61}
$$

We then obtain:

$$
\frac{\partial G}{\partial Z}=\begin{bmatrix}
-(\mu_H^\alpha+\sigma_H^\alpha+\kappa^\alpha) & \dfrac{\eta_1^\alpha}{\Omega_H}S_H+d\dfrac{\eta_1^\alpha}{\Omega_H}V_H & 0 & \dfrac{\eta_2^\alpha}{\Omega_H}S_H+d\dfrac{\eta_2^\alpha}{\Omega_H}V_H & \dfrac{\eta_3^\alpha}{\Omega_H}S_H+d\dfrac{\eta_3^\alpha}{\Omega_H}V_H\\
\sigma_H^\alpha & -(\mu_H^\alpha+\gamma_H^\alpha) & 0 & 0 & 0\\
0 & \dfrac{\eta_4^\alpha}{\Omega_A}S_A & -(\mu_A^\alpha+\sigma_A^\alpha) & \dfrac{\eta_5^\alpha}{\Omega_A}S_A & \dfrac{\eta_6^\alpha}{\Omega_A}S_A\\
0 & 0 & \sigma_A^\alpha & -(\mu_H^\alpha+\gamma_H^\alpha) & 0\\
0 & 0 & 0 & \rho^\alpha & -\omega^\alpha
\end{bmatrix}
\tag{62}
$$

$$A = \frac{\partial G(U^*,0)}{\partial Z} = \begin{bmatrix} -(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) & \Upsilon_1 & 0 & \Upsilon_2 & \Upsilon_3 \\ \sigma_H^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 & 0 & 0 \\ 0 & \eta_4^\alpha & -(\mu_A^\alpha + \sigma_A^\alpha) & \eta_5^\alpha & \eta_6^\alpha \\ 0 & 0 & \sigma_A^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 \\ 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix} \tag{63}$$

Where:

$$\begin{aligned} \Upsilon_1 &= \frac{\eta_1^\alpha \phi^\alpha + \eta_1^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha} \\ \Upsilon_2 &= \frac{\eta_2^\alpha \phi^\alpha + \eta_2^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha} \\ \Upsilon_3 &= \frac{\eta_3^\alpha \phi^\alpha + \eta_3^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha} \end{aligned} \tag{64}$$

$$G^*(U,Z) = AZ - G(U,Z) = \begin{bmatrix} (\eta_1^\alpha + \eta_2^\alpha + \eta_3^\alpha) I_H \left(1 - \frac{S_H + dV_H}{\Omega_H} + \mu_H^\alpha \kappa^\alpha (d-1)\right) \\ 0 \\ (\eta_4^\alpha + \eta_5^\alpha + \eta_6^\alpha) I_H \left(1 - \frac{S_A}{\Omega_A}\right) \\ 0 \\ 0 \end{bmatrix} \tag{65}$$

Since all parameters are positives also we have $\frac{S_H + dV_H}{\Omega_H} \ll 1$ and $\mu_H^\alpha \kappa^\alpha (d-1) \ll 1$.It's follows that $G1 \geq 0$, it's evident that $G3 \geq 0$.

Hence $G^*(U,Z) \geq 0 \quad \forall (U,Z) \in \Psi$.

Therefore the DFE point Φ_0 of the model (3) is globally assymptotically stable. End of the proof. □

3.7. Endemic Equilibrium Points EE.

Now we introduce the $(S_H, E_H, V_H, I_H, S_A, E_A, I_A, C_e) \in \mathbb{R}_+^8$ disease. The model has an concordance endemic equilibrium point shown by $E^* = (S_H^*, E_H^*, V_H^*, I_H^*, S_A^*, E_A^*, I_A^*, C_e^*)$.

The Endemic Equilibrium point is the solution of the $(S_H, E_H, V_H, I_H, S_A, E_A, I_A, C_e)$ model whose disease persist in the population of human, the population of animals and the environmental impact. We can calculate it well by equating each equation of the system (3) by zero.

Then

(66)
$$\begin{cases} \Lambda_H^\alpha + \phi^\alpha V_H^* - \left(\dfrac{\eta_1^\alpha I_H^* + \eta_2^\alpha I_A^* + \eta_3^\alpha C_e^*}{\Omega_H}\right) S_H^* - \kappa^\alpha \Lambda_H^\alpha - \mu_H^\alpha S_H^* & = 0, \\ \left(\dfrac{\eta_1^\alpha I_H^* + \eta_2^\alpha I_A^* + \eta_3^\alpha C_e^*}{\Omega_H}\right) S_H^* + d\left(\dfrac{\eta_1^\alpha I_H^* + \eta_2^\alpha I_A^* + \eta_3^\alpha C_e^*}{\Omega_H}\right) V_H^* - (\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) E_H^* & = 0, \\ \kappa^\alpha(\Lambda_H^\alpha + E_H^*) - (\mu_H^\alpha + \phi^\alpha) V_H^* - d\left(\dfrac{\eta_1^\alpha I_H^* + \eta_2^\alpha I_A^* + \eta_3^\alpha C_e^*}{\Omega_H}\right) V_H^* & = 0, \\ \sigma_H^\alpha E_H^* - (\mu_H^\alpha + \gamma_H^\alpha) I_H^* & = 0, \\ \Lambda_A^\alpha - \left(\dfrac{\eta_4^\alpha I_H^* + \eta_5^\alpha I_A^* + \eta_6^\alpha C_e^*}{\Omega_A}\right) S_A^* - \mu_A^\alpha S_A^* & = 0, \\ \left(\dfrac{\eta_4^\alpha I_H^* + \eta_5^\alpha I_A^* + \eta_6^\alpha C_e^*}{\Omega_A}\right) S_A^* - (\mu_A^\alpha + \sigma_A^\alpha) E_A^* & = 0, \\ \sigma_A^\alpha E_A^* - (\mu_A^\alpha + \gamma_A^\alpha) I_A^* & = 0, \\ \rho^\alpha I_A^* - \omega^\alpha C_e^* & = 0 \end{cases}$$

(67)
$$\begin{cases} S_H^* &= \dfrac{[\Lambda_H(1-\kappa^\alpha) + \phi^\alpha V_H^*]\Omega_H}{\mu_H^\alpha \Omega_H + (\eta_1 I_H^* + (\eta_2^\alpha + \eta_3^\alpha \rho^\alpha/\omega^\alpha) I_A^*)} \\ E_H^* &= \dfrac{\mu_H^\alpha + \gamma_H^\alpha}{\sigma_H \alpha} I_H^* \\ V_H^* &= \dfrac{\kappa^\alpha \Omega_H(\Lambda_H^\alpha \sigma_H^\alpha + \mu_H^\alpha + \gamma_H^\alpha)}{\Omega_H(\mu_H^\alpha + \phi^\alpha) + d(\eta_1 I_H^* + (\eta_2^\alpha + \eta_3^\alpha \rho^\alpha/\omega^\alpha) I_A^*)} \\ I_H^* &= \dfrac{(\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)\left[\Omega_A \mu_A^\alpha + (\eta_5^\alpha + \eta_6^\alpha \rho^\alpha/\omega^\alpha) I_A^*\right] - \Lambda_A^\alpha \sigma^\alpha(\eta_5^\alpha + \eta_5^\alpha \rho^\alpha/\omega_A^\alpha)}{\eta_4^\alpha \Lambda_H^\alpha \sigma_H^\alpha - \eta_4^\alpha(\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)} \\ S_A^* &= \dfrac{\Lambda_A}{\mu_A} - \dfrac{(\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)}{\mu_A^\alpha \sigma_A^\alpha} I_A^* \\ E_A^* &= \dfrac{\mu_A^\alpha + \gamma_A^\alpha}{\sigma_A^\alpha} I_A^* \\ I_A^* &= I_A^* \\ C_e^* &= \dfrac{\rho^\alpha}{\omega^\alpha} I_A^* \end{cases}$$

3.8. Global Stability of the Endemic Equilibrium Points: The global stability of the Endemic Equilibrium $E^* = (S_H^*, E_H^*, V_H^*, I_H^*, S_A^*, E_A^*, I_A^*, C_e^*)$ for the fractional order of the system model (3) is established following theorem as:

Theorem 3.5. *Let $\alpha \in (0,1]$, and $R_0 > 1$. Then the endemic equilibrium E of the proposed epidemic model* (3) *of fractional order model is globally stable in the interior of* Ψ.

Proof of theorem 3.5. To prove the global stability of the point E^*, we consider the Volterra-type Lyapunov functional approach [28] to define a function
$L(t) : \varepsilon(t) = [S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t)]^T \longrightarrow \mathbb{R}$, as

$$
\begin{aligned}
L(t) = & \frac{1}{a_1}(S_H - S_H^* - S_H^* \log \frac{S_H}{S_H^*}) + \frac{1}{a_2}(E_H - E_H^* - E_H^* \log \frac{E_H}{E_H^*}) \\
& + \frac{1}{a_3}(V_H - V_H^* - V_H^* \log \frac{V_H}{V_H^*}) + \frac{1}{a_4}(I_H - I_H^* - I_H^* \log \frac{I_H}{I_H^*}) \\
& + \frac{1}{a_5}(S_A - S_A^* - S_A^* \log \frac{S_A}{S_A^*}) + \frac{1}{a_6}(E_A - E_A^* - E_A^* \log \frac{E_A}{E_A^*}) \\
& + \frac{1}{a_7}(I_A - I_A^* - I_A^* \log \frac{I_A}{I_A^*}) + \frac{1}{a_8}(C_e - C_e^* - C_e^* \log \frac{C_e}{C_e^*})
\end{aligned} \tag{68}
$$

where

$$
\begin{aligned}
a_1 &= \lambda_H^\alpha + \mu_H^\alpha \\
a_2 &= \mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha \\
a_3 &= \mu_H^\alpha + \phi^\alpha + d\lambda_H^\alpha \\
a_4 &= \mu_H^\alpha + \gamma_H^\alpha \\
a_5 &= \lambda_A^\alpha + \mu_A^\alpha \\
a_6 &= \mu_A^\alpha + \sigma_A^\alpha \\
a_7 &= \mu_A^\alpha + \gamma_A^\alpha \\
a_8 &= \omega^\alpha
\end{aligned}
$$

The function L(t) is defined, continuous and positive definite for all $t \geq 0$. It can be verified that the equality holds if and only if $S_H = S_H^*, E_H = E_H^*, V_H = V_H^*, I_H = I_H^*, S_A = S_A^*, E_A = E_A^*, I_A = I_A^*, C_e = C_e^*$.

The α order of $L(S_H, E_H, V_H, I_H, S_A, E_A, I_A, C_e)$ is calculate to show $D_t^\alpha L \leq 0$ at the endemic equilibrium point.

$$
\begin{aligned}
D_t^{\alpha} L = {} & \frac{1}{a_1}\left(\frac{S_H - S_H^*}{S_H}\right) D_t^{\alpha} S_H + \frac{1}{a_2}\left(\frac{E_H - E_H^*}{E_H}\right) D_t^{\alpha} E_H + \frac{1}{a_3}\left(\frac{V_H - V_H^*}{V_H}\right) D_t^{\alpha} V_H \\
& + \frac{1}{a_4}\left(\frac{I_H - I_H^*}{I_H}\right) D_t^{\alpha} I_H + \frac{1}{a_5}\left(\frac{S_A - S_A^*}{S_A}\right) D_t^{\alpha} S_A + \frac{1}{a_6}\left(\frac{E_A - E_A^*}{E_A}\right) D_t^{\alpha} E_A \\
& + \frac{1}{a_7}\left(\frac{I_A - I_A^*}{S_A}\right) D_t^{\alpha} I_A + \frac{1}{a_8}\left(\frac{C_e - C_e^*}{C_e}\right) D_t^{\alpha} C_e
\end{aligned} \tag{69}
$$

By substituting, and on simplification using the endemic state condition of model (3), we have from Eq. (69) as:

$$
\begin{aligned}
D_t^{\alpha} L = {} & -\frac{(S_H - S_H^*)^2}{S_H} - \frac{(E_H - E_H^*)^2}{E_H} - \frac{(V_H - V_H^*)^2}{V_H} - \frac{(I_H - I_H^*)^2}{I_H} - \frac{(S_A - S_A^*)^2}{S_A} \\
& - \frac{(E_A - E_A^*)^2}{E_A} - \frac{(I_A - I_A^*)^2}{S_A} - \frac{(C_e - C_e^*)^2}{C_e}
\end{aligned} \tag{70}
$$

From the above calculation we can see that $D_t^{\alpha} L \leq 0$

We note that if $R_0 > 1$, then the right-hand side of Eq. (70) is negative and it is equal to zero if $S_H = S_H^*, E_H = E_H^*, V_H = V_H^*, I_H = I_H^*, S_A = S_A^*, E_A = E_A^*, I_A = I_A^*, C_e = C_e^*$.

According to the LaSalle's invariance principle [29, 30], and

[28], we know that all solutions in Ψ converge to E^*. Therefore, the endemic state of the model (3) is globally asymptotically stable when $R_0 > 1$ [31]. This completes the proof of (3.5). $\square$

TABLE 2. Sensitivity indices for R_0.

Parameter	**Index**
μ_A	-0.0779
μ_H	-0.1700
η_1	0.0332
η_4	0.1430
η_5	0.4223
η_6	0.2585
σ_A	0.0313
σ_H	0.1213
κ	-0.1254
α_A	-0.7772
α_H	-0.0979
ρ	0.2585
ϕ	-0.0571
d	0.0098
ω	-0.4015

TABLE 3. Descriptions and values of parameters in model.

Parameter	Value	Interpretation	Source
Λ_H	Recruitment rate into the susceptible human population	36	[4], [5]
Λ_A	Recruitment rate into the susceptible animal population	200	[5]
μ_A	Animal natural mortality rate	0.015	Estimated
μ_H	Human natural mortality rate	0.04	Estimated
η_1, η_2, η_3	Humans infection rate from I_H, I_A, and C_e, respectively	0.35, 0.55, 0.999	[5]
σ_A	Animal incubation period	0.38	Estimated
σ_H	Human incubation period	0.38	Estimated
κ	Human vaccination rate	0.8	Estimated
α_A	Animal disease-related death rate	0.25	Estimated
α_H	Human disease-related death rate	0.05	[4]
ρ	Dairy products production rate	0.6	Estimated
ϕ	Human loss rate of immunity	0.03	Estimated
d	The human efficacy of the vaccine	0.5	Estimated
ω	The decay rate in the contaminated environment	0.7	Estimated
η_4, η_5, η_6	Animals infection rate from I_H, I_A, and C_e, respectively	0.25, 0.7, 0.5	[5], Estimated, Estimated

4. NUMERICAL RESULTS AND DISCUSSION

4.1. The Basic reproduction number R_0 without vaccination. Let us denote R_0 without vaccination as R_0^*.

Using the parameters in table 3 and Maple software for computations, R_0^* and R_0 are given as follow:

$$R_0^* = \frac{R_1}{2} + \frac{\sqrt{R_2^2 + 4R_3}}{2}$$

where

$$R_1 = \frac{\beta_1 \sigma_H}{(\mu_H + \sigma_H)(\mu_H + \alpha_H)} + \frac{\beta_5 \sigma_A}{(\mu_A + \sigma_A)(\mu_A + \alpha_A)} + \frac{\beta_6 \rho\, \sigma_A}{(\mu_A + \alpha_A)(\mu_A + \sigma_A)\omega}$$

$$R_2 = \frac{\beta_1 \sigma_H}{(\mu_H + \sigma_H)(\mu_H + \alpha_H)} - \frac{\beta_5 \sigma_A}{(\mu_A + \sigma_A)(\mu_A + \alpha_A)} - \frac{\beta_6 \rho\, \sigma_A}{(\mu_A + \alpha_A)(\mu_A + \sigma_A)\omega}$$

$$R_3 = \frac{\sigma_A \mu_H (\beta_2 \omega + \beta_3 \rho) \beta_4 \sigma_H}{\mu_H (\mu_H + \sigma_H)(\mu_H + \alpha_H)(\mu_A + \sigma_A)(\mu_A + \alpha_A)\omega}$$

- $R_0^* = 7.4296$

- $R_0 = 4.9574$

From the above calculations, it indicates that the best way in minimizing the bovine tuberculosis is to use more vaccination in both human and animal populations.

4.1.1. *Herd Imminuty Threshold H_1:* We are therefore motivated to determine the number of people or animals that should receive vaccinations when R_0^* = 7.4296 based on the previously mentioned computations.

$$H_1 = 1 - \frac{1}{R_0^*} = 0.86$$

This shows that if $R_0^* = 7.4296$, then 86% of individuals and animals should receive vaccination.

4.2. Sensitivity Analysis of Basic Reproduction Number R_0. Understanding how each parameter affects the model output and its impact on the spread of disease throughout the population is made possible by the sensitivity analysis of R_0 [32]. Using the normalized forward sensitivity analysis index employed by Silva [33] and Torres [32], we undertake sensitivity analysis of R_0.

$$\Psi_\beta^{R_0} = \left(\frac{\partial R_0}{\partial \beta}\right)\left(\frac{\beta}{R_0}\right),$$

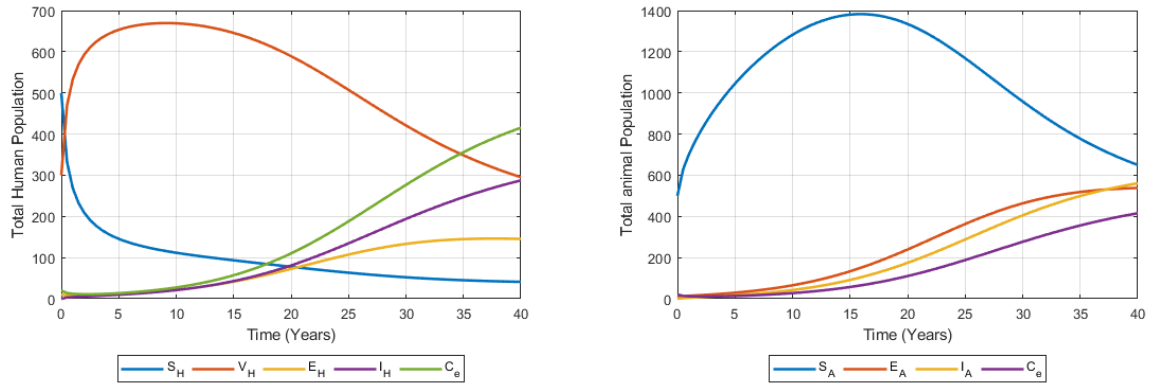
is the formula for the normalized forward sensitivity index of variable β with respect to the fundamental reproduction number R_0.

Table 2 lists the sensitivity index of each parameter to the fundamental reproduction number R_0 using estimated parameters and information from related literature.

According to sensitivity analysis, the evolution of bTB are driven by animal infection rates associated with the consumption of dairy products η_6 and contact rates with infectious animals η_5 as well as animal infection rates associated with the contact of infectious humains η_4, the animal and human incubation period, σ_A and σ_H respectively. The rate of making dairy products ρ is typically the most sensitive characteristic. The fundamental reproduction number R_0 increases by 0.018% for every 10% increase in dairy products. The fundamental reproduction number R_0 decreases as a result of an increase in the animal mortality rate owing to disease α_A, the animal natural mortality rate μ_A, the human disease-induced death rate α_H, the human natural mortality rate μ_H, the decay rate of dairy products ω, and the human vaccination rate.

We also note that, the human infection rates η_2 and η_3 from infectious animals and contaminated environment have no effect on the fundamental reproduction number R_0.

4.3. Numerical simulation. By taking into account the variables that influence the dynamics of bTB transmission, we address the evolution of bTB in the human and animal populations in this section. We use both estimated parameters and ones from the pertinent literature, as shown in Table 3 to illustrate the behavior of the model for different fractional order $1 < \alpha \leq 1$ and differents values for those parameters.

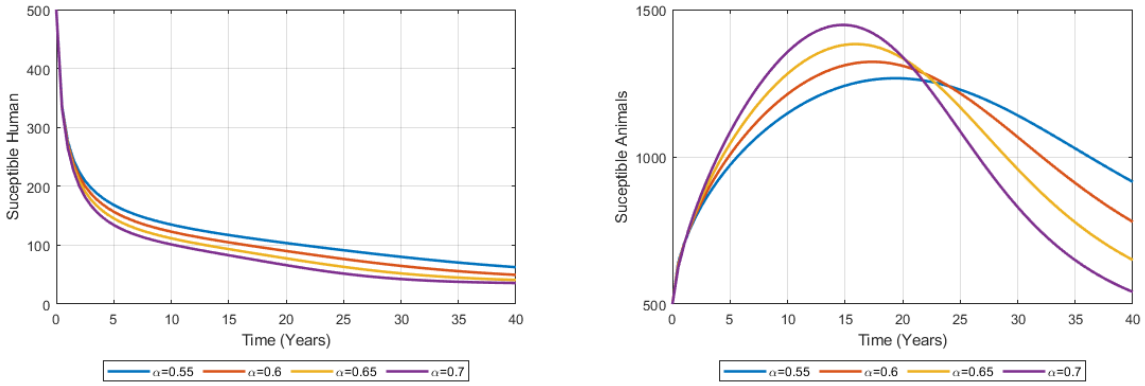


(A) (B)

FIGURE 2. Dynamics of bTB in human population (a) and animal population (b) for $\alpha = 65$.

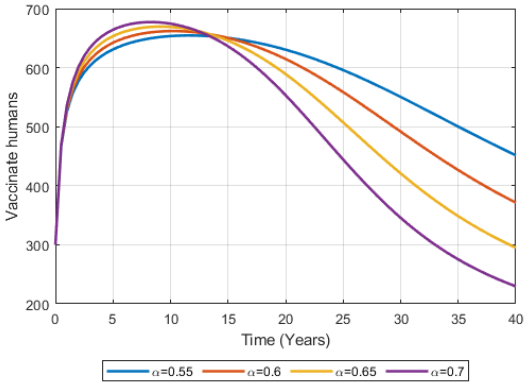
As seen in Figure 2, the number of susceptible people and animals decreases after contracting bTB from infected people and animals as well as after ingesting infected dairy products. But the people in the susceptible class decrease more than the case of animals, this is because of the vaccination for the human population. They both migrate into the exposed class and eventually into the infectious class.

Figures 3a, (3b), and (3c) show the effect of varying α on susceptible humans, susceptible animals and vaccinated humans respectively. The animal population is more infected than the human population as shown in figure 4, this can be explain by the fact that only humans receive vaccination.



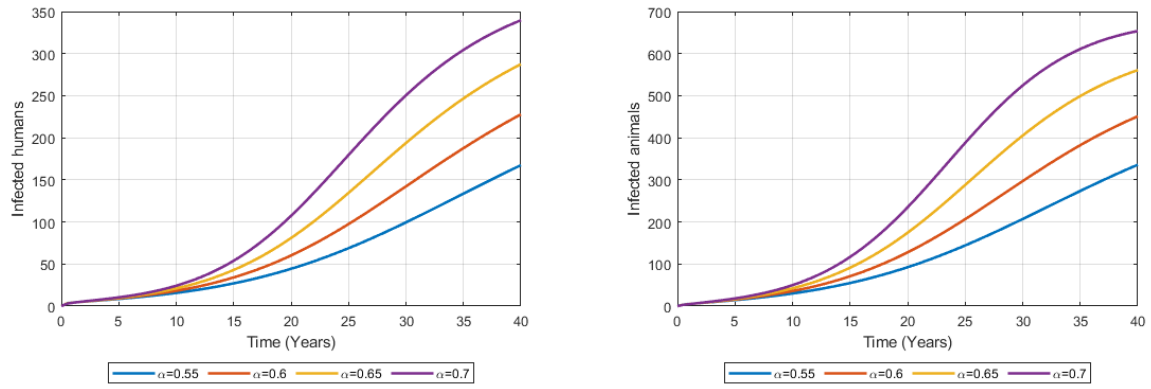
(A)

(B)



(C)

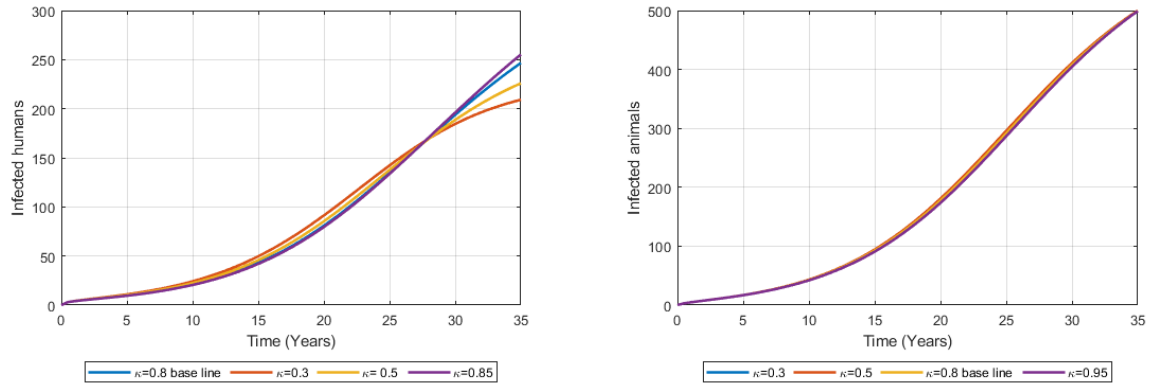
FIGURE 3. Variation of α for susceptible humans (a), animals (b), and vaccinated humans (c) population.



(A) (B)

FIGURE 4. Variation of α for infected humans (a) and infected animal(b) population.

4.3.1. *Impact of vaccination rate on infected humans and animals.*



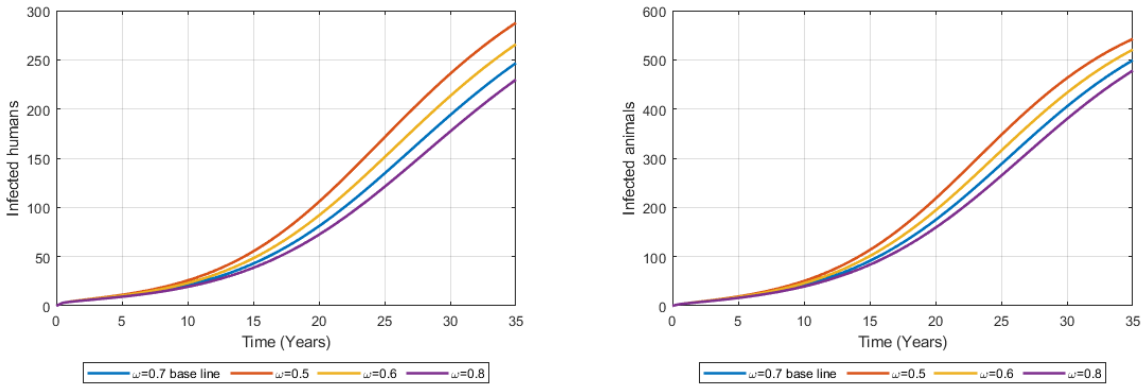
(A) (B)

FIGURE 5. Variation of κ for infected humans (a) and infected animal(b) population.

Figure 5 illustrates the outcomes of a numerical simulation carried out by varying the vaccination rate κ for human population while maintaining the other parameters constant. The simulation results clearly demonstrate that the plotted graphs show a downward trend as the vaccination rate κ increases for human population, but no significant effect for the animal population. This suggests that when the vaccination rate κ rises, the number of infected people decreases.

Consequently, it is crucial for the government and livestock farming experts to advise breeders to promptly vaccinate people and animals and put infected animals under quarantine as soon as they exhibit symptoms. By taking this measure, the spread of infection can be mitigated, leading to better human health and improved animal breeding outcomes.

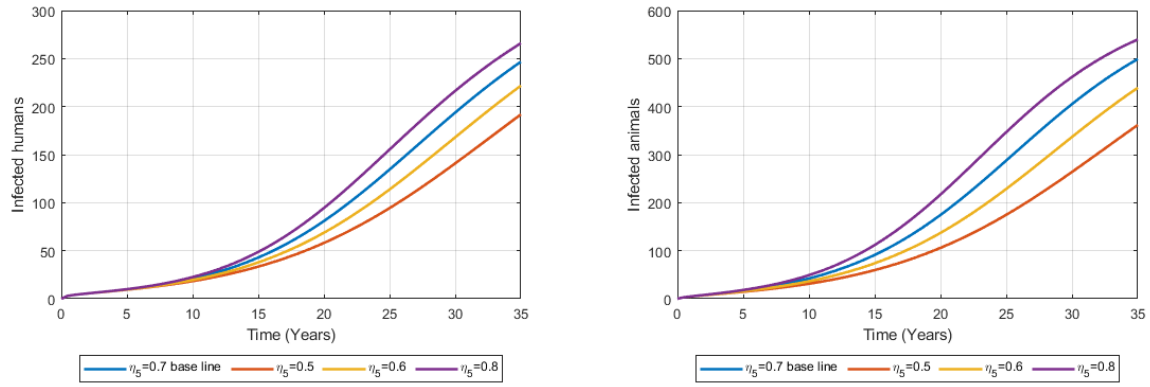
4.3.2. *Investigating the influence of the decay rate on the contaminated environment.* Figure 6 illustrates the outcomes of a numerical simulation carried out by varying the rate of decaying ω for contaminated environment (dairy products and meat) while maintaining the other parameters fixed. The findings demonstrate a clear correlation between the reduction of the decay rate and an increase of infectious humans and animals. Consequently, it can be inferred that elevating the decay rate significantly aids in eradicating the disease from both human and animal population.



(A) (B)

FIGURE 6. Variation of ω for infected humans (a) and infected animal (b) population.

4.3.3. *Impact of the animal infection rate from infected animals.*



(A) (B)

FIGURE 7. Variation of η_5 for infected humans (a) and infected animal(b) population .

The numerical result achieved by altering the animal infecion rate η_5 from infected animals while maintaining other parameters constant is shown in Figure 7. The quantity of infected animals and humans is increased after the value of η_5 is raised from 0.5 to 0.8. The proportion of diseased animals and humans are larger at $\eta_5 = 0.8$ than at other times. Overall, the numerical outcomes demonstrate that raising the animal infecion rate value causes an increase in the number of infected animals and humans. To stop the disease from spreading, all interested parties and policy makers must consider ways to reduce the animal infecion rate η_5 from infected animals by puting the infectious animals under quarantine.

5. Summery and Conclusion

We developed a fractional-order mathematical model in this study to simulate the progression of bovine tuberculosis in the presence of vaccination and a contaminated environment. The model was developed and described in Section (2). In Section (3), we looked at the qualitative behaviors of the model by finding the feasible region, the positivity of the solution, equilibrium points, and examining their local and global stability. We also looked at the fundamental reproduction number of the model. Through sensitivity analysis of the basic reproduction number, the traits that have a substantial impact on the management of bovine TB have been found. In Section (4), the results of the numerical simulation are examined. In this numerical simulation, we investigated the influence of the parameters κ, ω, and η_5 on the fractional order model. As a result of this analysis, we can draw the conclusion that increasing the vaccination rate κ of both the human and animal populations will greatly slow the spread of the bovine TB illness in both those populations. Accordingly, bovine TB management tries to reduce the disease's infection in both human and animal populations based on the study's findings. In this case, lowering the animal infection rate from infected animals, while increasing the decay rate of the polluted environment, and increasing the human population's vaccination rate, ought to aid in disease control.

Conflict of Interests

The authors declare that there is no conflict of interests.

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