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Research Article

Fractional-Order Model for Evolution of Bovine Tuberculosis with Vaccination and Contaminated Environment

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Bovine tuberculosis (bTB) is a zoonotic disease that is commonly transmitted via inhaling aerosols, drinking unpasteurized milk, and eating raw meat. We use a fractional-order model with the Caputo sense to examine the evolution of bovine tuberculosis transmission in human and animal populations, including a vaccine compartment for humans. We derived and obtained the threshold quantity R_0 to ascertain the illness state. We established conditions guaranteeing the asymptotic stability of the equilibria (locally and globally). Sensitivity analysis was conducted to identify the factors that govern the dynamics of tuberculosis. The study demonstrates that the rate of human-to-animal transmission of tuberculosis and environmental pollution and the rate of bTB transmission between animals all affect tuberculosis transmission. However, as vaccination rates increase and fewer individuals consume contaminated environment products (such as meat, milk, and other dairy products), the disease becomes less common in humans. To manage bovine TB, it is advised that information programmes be implemented, the environment be monitored, infected persons be treated, contaminated animals be vaccinated, and contaminated animals be quarantined. The usefulness of the discovered theoretical results is demonstrated through numerical experiments.

1. Introduction

There has been progress in the fight against tuberculosis (TB) in Africa, but several obstacles are hampering efforts to end this preventable and curable disease. Global targets to eradicate the illness by 2030 are increasingly improbable at the current rate [1]. Contact is the primary means of transmission for tuberculosis (TB), a chronic infectious disease mostly affecting the respiratory system. Reports [2] state that Africa has the highest prevalence of instances. India, China, and Indonesia followed with 72%, 27%, and 9%, respectively. The reorientation of funds to the COVID-19 response has hampered the provision of basic services in a number of countries. Due to the lockdowns, many who have tuberculosis had found it difficult to receive treatment. The ability to detect drug-resistant tuberculosis has been influenced by COVID-19 [1]. In comparison to 2019, the number of cases reported in the WHO African Region declined by 28% in 2020 [1].

Cattle tuberculosis is a zoonotic infectious disease that is classified as a class B animal epidemic by the OIE (Office International des Epizooties). Humans and other animals may contract an infection mostly from an infected animal. The respiratory and digestive systems are the primary pathways of transmission. Contact with ill animals or consumption of their raw milk can result in infection in both healthy humans and animals [3–5]. Since bTB-infected animals are put down as soon as they get ill, the disease has a significant negative economic impact [5]. In addition, bTB deteriorates health and can occasionally be lethal. Some people may lose

their self-employment as a result of it, especially those whose primary source of income is cattle rearing [6]. Bovine tuberculosis can transmit from cattle to humans through three primary routes: consuming raw meat, inhaling aerosols, and drinking unpasteurized milk [7]. Other ways that bTB spreads among animals include the consumption of contaminated milk, particularly during lactation, and breathing in of aerosols [8]. It can also spread through close contact between infected and uninfected animals.

The intradermal skin test is the most established and widely used approach for bTB diagnosis [9]. The fluctuating sensitivity and specificity are its principal drawbacks, as demonstrated in a number of papers. Furthermore, the use of tuberculosis vaccination procedures makes this test difficult because sensitised animals yield false-positive results [8]. A deterministic mathematical model is developed in [5] to explore the dynamics of bTB transmission in infected individuals as well as animals. To determine the disease's behaviour, the basic reproduction number R_0 is computed. According to the sensitivity analysis, the frequency at which dairy products are generated, animals infect other animals with bTB, and humans contract bTB from contaminated dairy products is what causes bTB to spread.

A meta-analysis by specialists from India, the USA, the UK, the Netherlands, and Ethiopia that assesses the impact of the BCG vaccination on cattle is an intriguing article [10]. According to their analyses, BCG immunization may hasten the control of bTB in endemic areas. Publications pertaining to the immunology of Mycobacterium bovis (Mb) infections have been written. The pathophysiology of tuberculosis in cattle is typified by lesions in the lymph nodes and lungs, which eventually lead to the development of granulomas. The immunopathology and chronic evolution of bTB are comparable to that of human TB in many aspects [8].

In [11], Ahmad et al. devised the reaction-diffusion model and applied the fractional differential equation to obtain traditional solutions to the nonlinear partial differential equation. The fraction differential calculus is a useful tool that may be used to explain the dynamics of many life events in the form of fractional orders. In [12], mathematical models for the evolution of potato leaf roll virus propagation are created using the differential equations with both integer and fractional orders. The models considered the combination of vectors and potato populations. The potato leaf roll virus (PLRV) model initially was created in integer order; however, the model was later extended into fractional order since fractional order gives memory and other benefits for modelling real-life occurrences.

In [3], the study looks at the evolution of bovine tuberculosis transmission in both human and animal populations, utilising a fractional-order model with the Caputo sense. The threshold quantity R_0 was also calculated using the Lyapunov functions of the Volterra type. In their study titled Review of Fractional Epidemic Models [13], concentrated on summarising several variants of the fractional epidemic model and assessing the outcomes of epidemiological modelling, specifically the fractional epidemic model. They developed simple, effective analytical techniques for solving fractional epidemic models that are easy to adapt and use. These techniques can assist the relevant organisations in managing, preventing, and even forecasting the spread of infectious diseases.

2. TB Model Description and Formulation

At any given time (t), the model divides total populations of humans and animals into seven (7) subpopulations (compartments), with contaminated environment C_e.

The following are bTB model assumptions: immigration and birth rates are within the susceptible human population, there exists a continuous interaction between human and animal populations, and because the model lacks a recovery class, it is presumed that there is no natural recovery. Consuming dairy and meat from sick animals can spread the illness to people.

The animal population size, Ω_A, is classified into three categories: susceptible (S_A), exposed (E_A), and infectious (I_A), where

$$S_A + E_A + I_A = \Omega_A. \tag{1}$$

The individual population size, denoted by Ω_H, is classified into vaccinated humans V_H, exposed E_H, infected I_H, and susceptible S_H.

$$S_H + V_H + E_H + I_H = \Omega_H. \tag{2}$$

2.1. Model Formulation. Table 1 shows bTB variables.

The bTB vulnerable population is recruited at an average rate of Λ_H. Furthermore, individuals catch the latent illness at a Λ_H rate by eating uncooked meat and milk and other dairy products from animals that are infected, along with regular coming into contact with contaminated people and livestock.

$$\lambda_H = \frac{\eta_1 I_H + \eta_2 I_A + \eta_3 C_e}{\Omega_H}. \tag{3}$$

Effective immunizations are administered to a selection of people at an average rate of κ, where $\kappa \in [0, 1]$. As the infectious stage advances, the graph shows that the passive infection of susceptible individuals S_H drops at a rate of γ_H and grows at a frequency of λ_H in the exposure class E_H. Human infections I_H rise at γ_H and fall at α_H due to disease-related death.

Natural death occurs in every human compartment at a rate of μ_H. Due to the decreasing influence of vaccine effectiveness with $(1 - d) \in [0, 1]$, vaccinated humans may transition to the exposure compartment at a rate of $d\lambda_H$. At a rate of ϕ, humans may become sensitive and lose their immunity.

Supplicant animals S_A are bred and transported into communities at an average of Λ_A; thereafter, they acquire a latent bovine TB infection through dairy consumption and interaction with ill humans and animals.

$$\lambda_A = \frac{\eta_4 I_H + \eta_5 I_A + \eta_6 C_e}{\Omega_A}. \tag{4}$$

Table 1: Parameter values and descriptions.

Parameter	Value	Interpretation	Sources
Λ_H	Human recruitment rate	37	[4, 5]
Λ_A	Animal recruitment rate	200	[5]
μ_A	Natural mortality rate of animals	0.015	Given
μ_H	Natural mortality rate of humans	0.04	Given
η_1, η_2, η_3	Infection rates in humans I_H, I_A, and C_e, respectively	0.350, 0.550, 0.999	[5]
σ_A	Incubation period for animals	0.3805	Given
σ_H	Incubation period for humans	0.3805	Given
κ	Vaccination rate in humans	0.805	Given
α_A	Death rate from animal sickness	0.2507	Given
α_H	Death rate from human sickness	0.0500	[4]
ρ	Rate of dairy production	0.600	[3]
ϕ	Human waning immunity rate	0.0300	Given
d	Vaccine efficacy rate	0.500	Given
ω	Environmental contamination rate	0.7	Given
η_4, η_5, η_6	Infection rate of animals from I_H, I_A, and C_e,	0.25, 0.70, 0.50	[5], given, given

Using the Caputo derivatives of order α, let us examine the fractional model where $0 < \alpha < 1$.

Using Diethelm's method [14], we will use the following system of fractional-order equations from Figure 1:

$$\begin{cases} {}_0^C D_t^\alpha S_H(t) = (1-\kappa^\alpha)\Lambda_H^\alpha + \phi^\alpha V_H - \left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) S_H - \mu_H^\alpha S_H, \\ {}_0^C D_t^\alpha E_H(t) = \left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) S_H + d\left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) V_H - (\mu_H^\alpha + \sigma_H^\alpha) E_H - \kappa^\alpha E_H, \\ {}_0^C D_t^\alpha V_H(t) = \kappa^\alpha \Lambda_H^\alpha + \kappa^\alpha E_H - (\mu_H^\alpha + \phi^\alpha) V_H - d\left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) V_H, \\ {}_0^C D_t^\alpha I_H(t) = \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha) I_H, \\ {}_0^C D_t^\alpha S_A(t) = \Lambda_A^\alpha - \left(\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A}\right) S_A - \mu_A^\alpha S_A, \\ {}_0^C D_t^\alpha E_A(t) = \left(\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A}\right) S_A - (\mu_A^\alpha + \sigma_A^\alpha) E_A, \\ {}_0^C D_t^\alpha I_A(t) = \sigma_A^\alpha E_A - (\mu_A^\alpha + \gamma_A^\alpha) I_A, \\ {}_0^C D_t^\alpha C_e(t) = \rho^\alpha I_A - \omega^\alpha C_e. \end{cases} \tag{5}$$

Under initial conditions,

$$S_H \geq 0, E_H \geq 0, I_H \geq 0, V_H \geq 0, S_A \geq 0, E_A \geq 0, \tag{6}$$

$I_A \geq 0, C_e \geq 0$ at $t = 0$.

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

Note: let us use the notation D^α instead of ${}_0^C D^\alpha$ in the rest of the discussion.

3. Model Analysis

3.1. bTB Invariant Region

Theorem 1. *Assume that* $\Psi = \{(S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t)) \in \mathbb{R}_+^8 : 0 \leq N_H \leq \Lambda_H/\mu_H \cup 0 \leq N_A \leq \Lambda_A/\mu_A \cup 0 \leq C_e \leq (\Lambda_A/\mu_A)(\rho/\omega)\}$.

Then, the model's system equation has a feasible solution set $\{(S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t))\}$, *which is bounded in the region* Ψ.

Proof.

(i) By summing the system equations of human population from model 3, the fractional derivative of the entire human population is as follows:

$$D^\alpha N_H = D^\alpha S_H + D^\alpha E_H + D^\alpha V_H + D^\alpha I_H, \tag{7}$$

$$D^\alpha N_H = \Lambda_H^\alpha + \phi^\alpha V_H - \lambda_H^\alpha S_H - \kappa^\alpha S_H - \mu_H^\alpha S_H + \lambda_H^\alpha S_H - (\mu_H^\alpha + \sigma_H^\alpha) E_H, \tag{8}$$

$$+\kappa^\alpha (S_H + E_H) - \mu_H^\alpha V_H + \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha) I_H, \tag{9}$$

$$D^\alpha N_H = \Lambda_H^\alpha - \mu_H^\alpha S_H - \mu_H^\alpha E_H - \mu_H^\alpha V_H - \mu_H^\alpha I_H - \gamma_H^\alpha V_H - \gamma_H^\alpha I_H, \tag{10}$$

$$D^\alpha N_H = \Lambda_H^\alpha - \mu_H^\alpha N_H - \gamma_H^\alpha I_H, \tag{11}$$

$$D^\alpha N_H \leq \Lambda_H^\alpha - \mu_H^\alpha N_H. \tag{12}$$

Note: compute without α on the right side in order to simplify the expressions. On both sides of Equation (12) using the Laplace transform [15],

$$\mathcal{L}\{D_t^\alpha N_H(t)\}(s) + \mathcal{L}\{\mu_H N_H(t)\}(s) \leq \mathcal{L}\{\Lambda_H\}(s). \tag{13}$$

On the LHS,

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

$$0 < \alpha < 1, \text{so } n = 1. \tag{14}$$

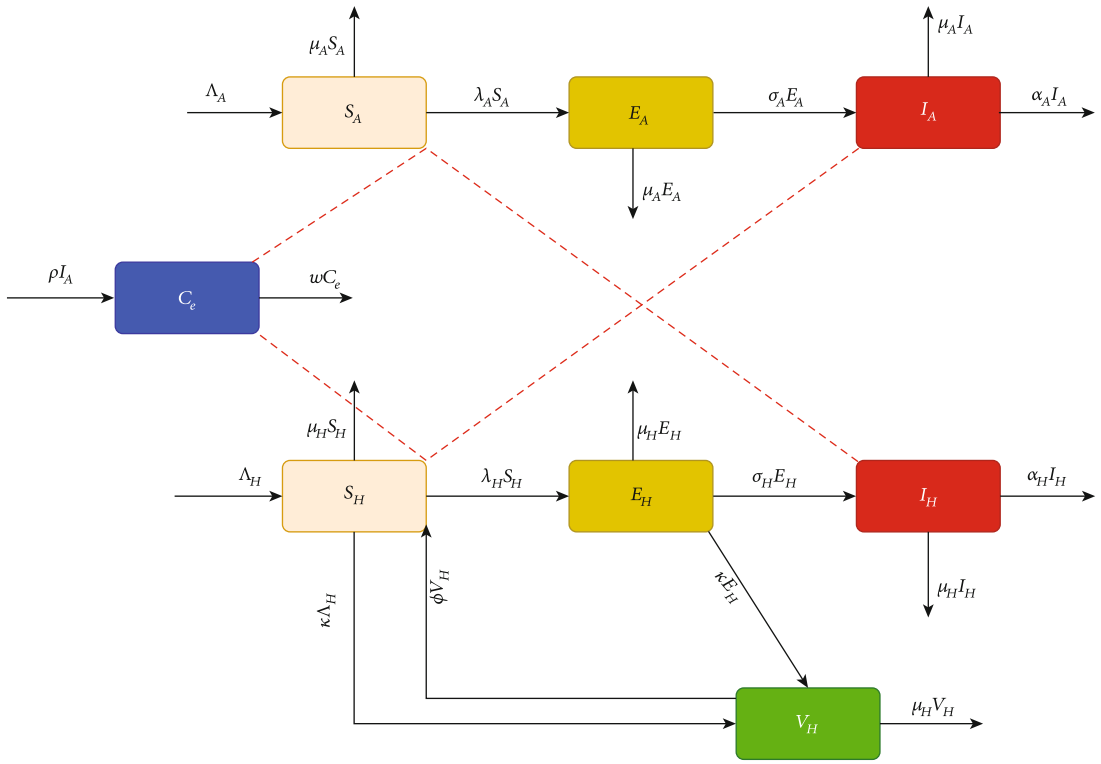


Figure 1: bTB model flow diagram.

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

$$\mathscr{L}\{\mu_H N_H(t)\}(s) = \mu_H \mathscr{N}_H(s). \tag{15}$$

Regarding the RHS,

$$\mathscr{L}\{\Lambda_H\}(s) = \Lambda_H \mathscr{L}\{1\} = \frac{\Lambda_H}{S}. \tag{16}$$

Equation (13) now looks like the following:

$$\mathscr{L}\{D_t^\alpha N_H(t)\}(s) + \mathscr{L}\{\mu_H N_H(t)\}(s) \le \mathscr{L}\{\Lambda_H\}(s), \tag{17}$$

$$s^\alpha \mathscr{N}_H(s) - s^{\alpha-1}N_H(0) + \mu_H \mathscr{N}_H(s) \le \frac{\Lambda_H}{s}, \tag{18}$$

$$\mathscr{N}_H(s)(s^\alpha + \mu_H) \le \frac{\Lambda_H}{s} + s^{\alpha-1}N_H(0). \tag{19}$$

Assuming that $s^{\alpha-1}N_H(0) = 0$ at $t = 0$ [12], then

$$\mathscr{N}_H(s) \le \Lambda_H \frac{s^{-1}}{s^\alpha + \mu_H}. \tag{20}$$

The Mittag-Leffler function and the inverse Laplace transform of $\mathscr{N}_H(s)$ are used; we obtain

$$\begin{aligned} N_H(t) &\le \Lambda_H \mathscr{L}^{-1}\left\{\frac{s^{-1}}{s^\alpha + \mu_H}\right\} \le \Lambda_H t^\alpha E_{\alpha,\alpha+1}(-\mu_H t^\alpha) \\ &\le \frac{\Lambda_H}{\mu_H}[1 - E_\alpha(-\mu_H t^\alpha)], \\ N_H(t) &\le \frac{\Lambda_H}{\mu_H}[1 - E_\alpha(-\mu_H t^\alpha)]. \end{aligned} \tag{21}$$

$\mu_H > 0$, and as $t \longrightarrow 0$, then $N_H(t) \longrightarrow \Lambda_H/\mu_H \ge 0$. Therefore,

$$0 \le N_H(t) \le \frac{\Lambda_H}{\mu_H},$$

$$\Psi_H = \left\{(S_H, E_H, V_H, I_H) \in \mathbb{R}_+^4 : S_H + E_H + V_H + I_H \le \frac{\Lambda_H^\alpha}{\mu_H^\alpha}\right\}. \tag{22}$$

(ii) Using the same methodology, the animal population will yield

$$\Psi_A = \left\{(S_A, E_A, I_A) \in \mathbb{R}_+^3 : S_H + E_H + I_H \le \frac{\Lambda_A^\alpha}{\mu_A^\alpha}\right\}. \tag{23}$$

(iii) The contaminated environment will use $0 < I_A \le \Lambda_A^\alpha/\mu_A^\alpha$.

The 8th equation of model (3) yields the following:

$$D^{\alpha}C_{e}(t) \leq \rho^{\alpha}\frac{\Lambda_{A}^{\alpha}}{\mu_{A}^{\alpha}} - \omega^{\alpha}C_{e}. \tag{24}$$

Using the equality case and performing the Laplace transform of Equation (24) on both sides, we have

$$\mathscr{L}\{D_{t}^{\alpha}C_{e}(t)\}(s) \leq \mathscr{L}\left\{\left(\rho^{\alpha}\frac{\Lambda_{A}^{\alpha}}{\mu_{A}^{\alpha}}\right) - \omega^{\alpha}C_{e}(t)\right\}(s). \tag{25}$$

Using the identical calculus method as the human population example, we obtain the following:

On the LHS,

$$\mathscr{L}\{D_{t}^{\alpha}C_{e}(t)\}(s) = s^{\alpha}\mathscr{C}_{e}(s) - s^{\alpha-1}C_{e}(0). \tag{26}$$

On the RHS,

$$\begin{aligned}\mathscr{L}\left\{\left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right) - \omega C_{e}(t)\right\}(s) &= \left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right)\mathscr{L}\{1\} - \omega\mathscr{L}\{C_{e}(t)\} \\ &= \frac{(\rho(\Lambda_{A}/\mu_{A}))}{S} - \omega\mathscr{C}_{e}(s).\end{aligned} \tag{27}$$

Now, Equation (28) becomes

$$\mathscr{C}_{e}(s) = \left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right)\frac{s^{-1}}{s^{\alpha}+\omega} + \frac{s^{\alpha-1}}{s^{\alpha}+\omega}C_{e}(0). \tag{28}$$

Taking $s^{\alpha-1}C_{e}(0) = 0$ at $t = 0$, then

$$\mathscr{C}_{e}(s) = \left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right)\frac{s^{-1}}{s^{\alpha}+\omega}. \tag{29}$$

Using (29)'s inverse Laplace transform, we obtain

$$\begin{aligned}C_{e}(t) &= \left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right)\mathscr{L}^{-1}\left\{\frac{s^{-1}}{s^{\alpha}+\omega}\right\} = \left(\rho\frac{\Lambda_{A}}{\mu_{A}}\right)t^{\alpha}E_{\alpha,\alpha+1}(-\omega t^{\alpha}), \\ C_{e}(t) &\leq \frac{\Lambda_{A}}{\mu_{A}}\frac{\rho}{\omega}[1 - E_{\alpha}(-\omega t^{\alpha})].\end{aligned} \tag{30}$$

$\omega > 0$ and, as $t \longrightarrow 0$, then $C_{e}(t) \longrightarrow (\Lambda_{A}/\mu_{A})(\rho/\omega) \geq 0$. Therefore,

$$0 \leq C_{e}(t) \leq \frac{\Lambda_{A}}{\mu_{A}}\frac{\rho}{\omega}, \tag{31}$$

and so

$$\Psi_{C_{e}} = \left\{C_{e} \in \mathbb{R}_{+} : C_{e} \leq \frac{\Lambda_{A}^{\alpha}}{\mu_{A}^{\alpha}}\frac{\rho^{\alpha}}{\omega^{\alpha}}\right\}. \tag{32}$$

Given a system of fractal-order equations in (5), the viable region is as follows:

$$\Psi \subset \mathbb{R}_{+}^{4} \times \mathbb{R}_{+}^{3} \times \mathbb{R}_{+}. \tag{33}$$

That comprises a set of positive invariants. □

This exhibits the model solution's boundedness.

3.2. Positivity. Since the initial values of each model equation are positive, then all of the model equations' solutions (5) remain positive for future times.

Lemma 2 (see [16]). *Assuming that $k(t) \in C[x, y]$ and ${}_{0}^{C}D_{t}^{\alpha}k(t) \in C[x, y]$ for $0 < \alpha \leq 1$, then*

$$k(t) = k(x) + \frac{1}{\Gamma(\alpha)}{}_{0}^{C}D_{t}^{\alpha}k(\varepsilon).(t-x)^{\alpha}, \tag{34}$$

where $x \leq \varepsilon \leq t, \forall t \in (x, y]$.

Remark 3. Consider $k(t) \in C[x, y]$ and ${}_{0}^{C}D_{t}^{\alpha}k(t) \in C[x, y]$ for $0 < \alpha \leq 1$. It follows from Lemma (2) that if ${}_{0}^{C}D_{t}^{\alpha}k(t) \geq 0$, $\forall t \in (x, y)$, then $k(t)$ increases for $\forall t \in [x, y]$, and if ${}_{0}^{C}D_{t}^{\alpha}k(t) \leq 0, \forall t \in (x, y)$, thus $k(t)$ decreases for $\forall t \in [x, y]$.

Theorem 4. *Let $S_{H}(0), E_{H}(0), V_{H}(0), I_{H}(0), S_{A}(0), E_{A}(0), I_{A}(0), C_{e}(0)$ be nonnegatives; 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 nonnegatives for all time $t > 0$.*

Proof. Taking all of the model's equations in Equation (5) at $t = 0$, we obtain

$${}_{0}^{C}D_{t}^{\alpha}S_{H}\Big|_{S_{H}=0} = (1-\kappa^{\alpha})\Lambda_{H}^{\alpha} + \phi^{\alpha}V_{H} \geq 0, \tag{35}$$

$${}_{0}^{C}D_{t}^{\alpha}E_{H}\Big|_{E_{H}=0} = \lambda_{H}S_{H} + d\lambda_{H}V_{H} \geq 0, \tag{36}$$

$${}_{0}^{C}D_{t}^{\alpha}V_{H}\Big|_{V_{H}=0} = \kappa^{\alpha}\Lambda_{H}^{\alpha} + \kappa^{\alpha}E_{H} \geq 0, \tag{37}$$

$${}_{0}^{C}D_{t}^{\alpha}I_{H}\Big|_{I_{H}=0} = \sigma_{H}^{\alpha}E_{H} \geq 0, \tag{38}$$

$${}_{0}^{C}D_{t}^{\alpha}S_{A}\Big|_{S_{A}=0} = \Lambda_{A}^{\alpha} > 0, \tag{39}$$

$${}_{0}^{C}D_{t}^{\alpha}E_{A}\Big|_{E_{A}=0} = \lambda_{A}S_{A} \geq 0, \tag{40}$$

$${}_{0}^{C}D_{t}^{\alpha}I_{A}\Big|_{I_{A}=0} = \sigma_{A}^{\alpha}E_{A} \geq 0, \tag{41}$$

$${}_{0}^{C}D_{t}^{\alpha}C_{e}\Big|_{C_{e}=0} = \rho^{\alpha}I_{A} \geq 0. \tag{42}$$

□

The solution $(S_{H}(t), E_{H}(t), V_{H}(t), I_{H}(t), S_{A}(t), E_{A}(t), I_{A}(t), C_{e}(t))$ cannot escape from the hyperplanes of $U(t) = 0$, $\forall U \in \Psi$ and for $t > 0$, because $S_{H}(0), E_{H}(0), V_{H}(0), I_{H}(0),$

$S_A(0), E_A(0), I_A(0), C_e(0)$ are nonnegatives, as per Equations (35)–(42) and by using Remark (3). Consequently, for all $t > 0$, all of the model's solutions with initial conditions in the set Ψ stay in Ψ. This area is a positive invariant set as a result.

3.3. Disease-Free Equilibrium (DFE). According to Φ_0, this is reached when there exists no disease in both the animal and human populations as a whole.

$$\begin{cases} {}_0^C D_t^\alpha S_H(t) = 0, \\ {}_0^C D_t^\alpha E_H(t) = 0, \\ {}_0^C D_t^\alpha V_H(t) = 0, \\ {}_0^C D_t^\alpha I_H(t) = 0, \\ {}_0^C D_t^\alpha S_A(t) = 0, \\ {}_0^C D_t^\alpha E_A(t) = 0, \\ {}_0^C D_t^\alpha I_A(t) = 0, \\ {}_0^C D_t^\alpha C_e(t) = 0. \end{cases} \tag{43}$$

Following some math, we obtain

$$\Phi_0 = \left(\frac{\Lambda_H^\alpha(\phi^\alpha + (1-\kappa^\alpha)\mu_H^\alpha)}{\mu_H(\mu_H^\alpha + \phi^\alpha)}, 0, \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}, 0, \frac{\Lambda_A^\alpha}{\mu_A^\alpha}, 0, 0, 0 \right). \tag{44}$$

3.4. The Basic Reproduction Number. We use the next-generation matrix algorithm to generate the bTB fundamental reproduction number R_0, as described in [17, 18].

$$\begin{cases} {}_0^C D_t^\alpha E_H(t) = \lambda_H^\alpha S_H + d\lambda_H^\alpha V_H - (\mu_H^\alpha + \sigma_H^\alpha)E_H - \kappa^\alpha E_H, \\ {}_0^C D_t^\alpha I_H(t) = \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha)I_H, \\ {}_0^C D_t^\alpha E_A(t) = \lambda_A^\alpha S_A - (\mu_A^\alpha + \sigma_A^\alpha)E_A, \\ {}_0^C D_t^\alpha I_A(t) = \sigma_A^\alpha E_A - (\mu_A^\alpha + \gamma_A^\alpha)I_A, \\ {}_0^C D_t^\alpha C_e(t) = \rho^\alpha I_A - \omega^\alpha C_e. \end{cases} \tag{45}$$

Let F_i represent the total quantity of newly acquired infections entering the system and V_i represent the number of infections leaving the system owing to births or deaths.

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

$$V_i = \begin{bmatrix} (\mu_H^\alpha + \sigma_H^\alpha)E_H + \kappa^\alpha E_H \\ -\sigma_H^\alpha E_H + (\mu_H^\alpha + \gamma_H^\alpha)I_H \\ (\mu_A^\alpha + \sigma_A^\alpha)E_A \\ -\sigma_A^\alpha E_A + (\mu_A^\alpha + \gamma_A^\alpha)I_A \\ -\rho^\alpha I_A + \omega^\alpha C_e \end{bmatrix}. \tag{46}$$

Denote the Jacobian matrices of F_i and V_i by F and V, respectively.

$$F = \begin{bmatrix} \frac{\partial F_1}{E_H} & \frac{\partial F_1}{I_H} & \frac{\partial F_1}{E_A} & \frac{\partial F_1}{I_A} & \frac{\partial F_1}{C_e} \\ \frac{\partial F_2}{E_H} & \frac{\partial F_2}{I_H} & \frac{\partial F_2}{E_A} & \frac{\partial F_2}{I_A} & \frac{\partial F_2}{C_e} \\ \frac{\partial F_3}{E_H} & \frac{\partial F_3}{I_H} & \frac{\partial F_3}{E_A} & \frac{\partial F_3}{I_A} & \frac{\partial F_3}{C_e} \\ \frac{\partial F_4}{E_H} & \frac{\partial F_4}{I_H} & \frac{\partial F_4}{E_A} & \frac{\partial F_4}{I_A} & \frac{\partial F_4}{C_e} \\ \frac{\partial F_5}{E_H} & \frac{\partial F_5}{I_H} & \frac{\partial F_5}{E_A} & \frac{\partial F_5}{I_A} & \frac{\partial F_5}{C_e} \end{bmatrix}$$

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

$$V = \begin{bmatrix} \frac{\partial V_1}{E_H} & \frac{\partial V_1}{I_H} & \frac{\partial V_1}{E_A} & \frac{\partial V_1}{I_A} & \frac{\partial V_1}{C_e} \\ \frac{\partial V_2}{E_H} & \frac{\partial V_2}{I_H} & \frac{\partial V_2}{E_A} & \frac{\partial V_2}{I_A} & \frac{\partial V_2}{C_e} \\ \frac{\partial V_3}{E_H} & \frac{\partial V_3}{I_H} & \frac{\partial V_3}{E_A} & \frac{\partial V_3}{I_A} & \frac{\partial V_3}{C_e} \\ \frac{\partial V_4}{E_H} & \frac{\partial V_4}{I_H} & \frac{\partial V_4}{E_A} & \frac{\partial V_4}{I_A} & \frac{\partial V_4}{C_e} \\ \frac{\partial V_5}{E_H} & \frac{\partial V_5}{I_H} & \frac{\partial V_5}{E_A} & \frac{\partial V_5}{I_A} & \frac{\partial V_5}{C_e} \end{bmatrix}$$

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

$$V^{-1} = \begin{bmatrix} \frac{1}{(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha)} & 0 & 0 & 0 & 0 \\ \frac{\sigma_H^\alpha}{(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha)(\mu_H^\alpha + \gamma_H^\alpha)} & \frac{1}{(\mu_H^\alpha + \gamma_H^\alpha)} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\mu_A^\alpha + \sigma_A^\alpha)} & 0 & 0 \\ 0 & 0 & \frac{\sigma_A^\alpha}{(\mu_A^\alpha + \sigma_A^\alpha)(\mu_A^\alpha + \gamma_A^\alpha)} & \frac{1}{(\mu_A^\alpha + \gamma_A^\alpha)} & 0 \\ 0 & 0 & \frac{\rho^\alpha \sigma_A^\alpha}{(\mu_A^\alpha + \sigma_A^\alpha)(\mu_A^\alpha + \gamma_A^\alpha)\omega^\alpha} & \frac{\rho^\alpha}{(\mu_A^\alpha + \gamma_A^\alpha)\omega^\alpha} & \frac{1}{\omega^\alpha} \end{bmatrix}. \tag{47}$$

$R_0 = FV^{-1}$. Let

$$A_1 = \frac{\eta_1 \sigma_H (dV_H + S_H)}{(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)},$$

$$A_2 = \frac{dV_H \eta_1 + \eta_1 S_H}{\mu_H + \gamma_H},$$

$$A_3 = \frac{(dV_H \eta_2 + \eta_2 S_H)\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)} + \frac{(dV_H \eta_3 + \eta_3 S_H)\rho\,\sigma_A}{(\mu_A + \gamma_A)(\mu_A + \sigma_A)\omega},$$

$$A_4 = \frac{dV_H \eta_2 + \eta_2 S_H}{\mu_A + \gamma_A} + \frac{(dV_H \eta_3 + \eta_3 S_H)\rho}{(\mu_A + \gamma_A)\omega},$$

$$A_5 = \frac{d\eta_3 V_H + \eta_3 S_H}{\omega},$$

$$B_1 = \frac{\eta_4 \sigma_H S_A}{(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)},$$

$$B_2 = \frac{\eta_4 S_A}{\mu_H + \gamma_H},$$

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

$$B_4 = \frac{\eta_5 S_A}{\mu_A + \gamma_A} + \frac{\eta_6 S_A \rho}{(\mu_A + \gamma_A)\omega},$$

$$B_5 = \frac{\eta_6 S_A}{\omega}. \tag{48}$$

Compute the eigenvalues of FV^{-1} and select the dominant one.

X is the eigenvalue of the matrix.

$$\begin{vmatrix} A_1 - X & A_2 & A_3 & A_4 & A_5 \\ 0 & -X & 0 & 0 & 0 \\ B_1 & B_2 & B_3 - X & B_4 & B_5 \\ 0 & 0 & 0 & -X & 0 \\ 0 & 0 & 0 & 0 & -X \end{vmatrix} = 0. \tag{49}$$

Equation (49) can be translated as follows:

$$X^3 \begin{vmatrix} A_1 - X & A_3 \\ B_1 & B_3 - X \end{vmatrix} = 0. \tag{50}$$

Characteristic equation is given as

$$X^3 \left[X^2 - (A_1 + B_3)X + A_1 B_3 - A_3 B_1\right] = 0. \tag{51}$$

Therefore, the highest eigenvalue is

$$X = \frac{A_1 + B_3}{2} + \frac{\sqrt{(A_1 - B_3)^2 + 4A_3 B_1}}{2}. \tag{52}$$

Evaluating and substituting A_1, A_3, B_1, and B_3 at the DFE Φ_0, we obtain

$$R_1 = A_1 + B_3 = \frac{\eta_1 \sigma_H (d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H)}{(\mu_H + \phi)(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)} + \frac{\sigma_A (\eta_5 \omega + \eta_6 \rho)}{(\mu_A + \sigma_A)(\mu_A + \gamma_A)\omega}, \tag{53}$$

$$R_2 = A_1 - B_3 = \frac{\eta_1 \sigma_H (d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H)}{(\mu_H + \phi)(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)} - \frac{\sigma_A (\eta_5 \omega + \eta_6 \rho)}{(\mu_A + \sigma_A)(\mu_A + \gamma_A)\omega}, \tag{54}$$

$$R_3 = A_3 B_1 = \frac{\eta_4 \sigma_H \sigma_A (d\kappa \mu_H - \kappa \mu_H + \phi + \mu_H)(\omega \eta_2 + \rho \eta_3)}{\omega(\mu_H + \phi)(\mu_A + \sigma_A)(\mu_A + \gamma_A)(\mu_H + \sigma_H + \kappa)(\mu_H + \gamma_H)}, \tag{55}$$

$$R_0 = \frac{R_1}{2} + \frac{\sqrt{R_2^2 + 4R_3}}{2}. \tag{56}$$

The terms $1/(\mu_H + \sigma_H + \kappa)$ and $1/(\mu_A + \sigma_A)$ in Equation (53) represent the mean amount of each humans and animals in their respective exposed compartment, and $1/(\mu_H + \phi)$ denotes the mean duration of time that each individual is exposed to in their respective classes, $1/(\mu_H + \gamma_H)$ denotes the mean ratio of time spent by each human in the vaccinated class, and $1/(\mu_A + \gamma_A)$ denotes the mean ratio of time each infectious populace devotes time to their infectious compartment.

$$\frac{\eta_1 \sigma_H [\phi + \mu_H (1 + d\kappa - \kappa)]}{(\mu_H + \phi)(\mu_H + \gamma_H)(\mu_H + \sigma_H + \kappa)}. \tag{57}$$

The proportion of diseased animals is represented by $(\sigma_A(\omega\eta_5 + \rho\eta_6))/(\omega(\mu_A + \gamma_A)(\mu_A + \sigma_A))$.

Equation (54) gives the sum of infected bTB through contact with diseased animals through ingestion of dairy products.

3.5. Analysis of Local Stability for DFE. If the eigenvalues of the Jacobian matrix are negative or have a negative real portion, DFE is considered locally asymptotically stable [5].

Theorem 5. *If all of the $J(\Phi_0)$ eigenvalues satisfy the condition that $|\arg \lambda_j| > \alpha\pi/2$, with $j = 1, 2, 3 \cdots$ and $0 < \alpha \leq 1$, Φ_0 is locally asymptotical and it is stable.*

Proof. Evaluating the Jacobian at DFE Φ_0, we end up with

$$J(\Phi_0) = \begin{bmatrix} -\mu_H^\alpha & 0 & \phi^\alpha & \eta_1^\alpha & 0 & 0 & -\eta_2^\alpha & -\eta_3^\alpha \\ 0 & -\mu_H^\alpha - \sigma_H^\alpha - \kappa^\alpha & 0 & d\eta_1^\alpha & 0 & 0 & d\eta_2^\alpha & d\eta_3^\alpha \\ 0 & \kappa^\alpha & -\mu_H^\alpha - \phi^\alpha & -d\dfrac{\eta_1^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} & 0 & 0 & -d\dfrac{\eta_2^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} & -d\dfrac{\eta_3^\alpha \kappa^\alpha}{\mu_H^\alpha + \phi^\alpha} \\ 0 & \sigma_H^\alpha & 0 & -\mu_H^\alpha - \gamma_H^\alpha & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\eta_4^\alpha & -\mu_A^\alpha & 0 & -\eta_5^\alpha & -\eta_6^\alpha \\ 0 & 0 & 0 & \eta_4^\alpha & 0 & -\mu_A - \sigma_A & \eta_5^\alpha & \eta_6^\alpha \\ 0 & 0 & 0 & 0 & 0 & \sigma_A^\alpha & -\mu_A^\alpha - \gamma_A^\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix}. \tag{58}$$

Matrix (58) has negative eigenvalues $-\mu_H^\alpha$, $-\mu_A^\alpha$, and $-\mu_H^\alpha - \phi^\alpha$, and those three eigenvalues satisfy the condition $|\arg \lambda_j| > \alpha\pi/2$ for all $0 < \alpha \leq 1$.

Matrix (58) reduces now to

$$R=\begin{bmatrix} -\mu_{\mathrm{H}}^{\alpha}-\sigma_{\mathrm{H}}^{\alpha}-\kappa^{\alpha} & d\eta_1^{\alpha} & 0 & d\eta_2^{\alpha} & d\eta_3^{\alpha} \\ \sigma_{\mathrm{H}}^{\alpha} & -\mu_{\mathrm{H}}^{\alpha}-\gamma_{\mathrm{H}}^{\alpha} & 0 & 0 & 0 \\ 0 & \eta_4^{\alpha} & -\mu_{\mathrm{A}}-\sigma_{\mathrm{A}} & \eta_5^{\alpha} & \eta_6^{\alpha} \\ 0 & 0 & \sigma_{\mathrm{A}}^{\alpha} & -\mu_{\mathrm{A}}^{\alpha}-\gamma_{\mathrm{A}}^{\alpha} & 0 \\ 0 & 0 & 0 & \rho^{\alpha} & -\omega^{\alpha} \end{bmatrix}. \tag{59}$$

If $\det(R)>0$ and $tr(R)=0$, thus DFE is locally stable. Equation (60) gives the trace of matrix R.

$$\begin{aligned} tr(R) = -((&(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})+(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})+(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}}) \\ &+(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})+\omega^{\alpha})<0. \end{aligned} \tag{60}$$

R has a determinant that is provided by

$$\begin{aligned} \det(R) = &-(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})\begin{vmatrix} -\mu_{\mathrm{H}}^{\alpha}-\gamma_{\mathrm{H}}^{\alpha} & 0 & 0 & 0 \\ \eta_4^{\alpha} & -\mu_{\mathrm{A}}-\sigma_{\mathrm{A}} & \eta_5^{\alpha} & \eta_6^{\alpha} \\ 0 & \sigma_{\mathrm{A}}^{\alpha} & -\mu_{\mathrm{A}}^{\alpha}-\gamma_{\mathrm{A}}^{\alpha} & 0 \\ 0 & 0 & \rho^{\alpha} & -\omega^{\alpha} \end{vmatrix} \\ &-\sigma_{\mathrm{H}}^{\alpha}\begin{vmatrix} d\eta_1^{\alpha} & 0 & d\eta_2^{\alpha} & d\eta_3^{\alpha} \\ \eta_4^{\alpha} & -\mu_{\mathrm{A}}-\sigma_{\mathrm{A}} & \eta_5^{\alpha} & \eta_6^{\alpha} \\ 0 & \sigma_{\mathrm{A}}^{\alpha} & -\mu_{\mathrm{A}}^{\alpha}-\gamma_{\mathrm{A}}^{\alpha} & 0 \\ 0 & 0 & \rho^{\alpha} & -\omega^{\alpha} \end{vmatrix} \\ = &(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})[(\sigma_{\mathrm{A}}^{\alpha}(\omega^{\alpha}\eta_5^{\alpha}+\rho^{\alpha}\eta_6^{\alpha}) \\ &-(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha}]+\sigma_{\mathrm{H}}^{\alpha}d\eta_1^{\alpha}(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha} \\ &+d\sigma_{\mathrm{H}}^{\alpha}\sigma_{\mathrm{A}}^{\alpha}[\omega^{\alpha}(\eta_2^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_5^{\alpha})+\rho^{\alpha}(\eta_3^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_6^{\alpha})]. \end{aligned} \tag{61}$$

Assume $\det(R)=0$; thus,

$$\begin{aligned} 0 = &\sigma_{\mathrm{A}}^{\alpha}(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})(\omega^{\alpha}\eta_5^{\alpha}+\rho^{\alpha}\eta_6^{\alpha}) \\ &-(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha} \\ &+\sigma_{\mathrm{H}}^{\alpha}d\eta_1^{\alpha}(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha}+d\sigma_{\mathrm{H}}^{\alpha}\sigma_{\mathrm{A}}^{\alpha} \\ &\cdot[\omega^{\alpha}(\eta_2^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_5^{\alpha})+\rho^{\alpha}(\eta_3^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_6^{\alpha})] \\ = &\frac{\sigma_{\mathrm{A}}^{\alpha}(\omega^{\alpha}\eta_5^{\alpha}+\rho^{\alpha}\eta_6^{\alpha})}{(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})}+\frac{\sigma_{\mathrm{H}}^{\alpha}d\eta_1^{\alpha}\omega^{\alpha}}{(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})} \\ &+\frac{d\sigma_{\mathrm{H}}^{\alpha}\sigma_{\mathrm{A}}^{\alpha}[\omega^{\alpha}(\eta_2^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_5^{\alpha})+\rho^{\alpha}(\eta_3^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_6^{\alpha})]}{(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha}}-1. \end{aligned} \tag{62}$$

Thus, $\det(R)>0$ if

$$\begin{aligned} &\frac{\sigma_{\mathrm{A}}^{\alpha}(\omega^{\alpha}\eta_5^{\alpha}+\rho^{\alpha}\eta_6^{\alpha})}{(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})}+\frac{\sigma_{\mathrm{H}}^{\alpha}d\eta_1^{\alpha}\omega^{\alpha}}{(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})} \\ &\quad+\frac{d\sigma_{\mathrm{H}}^{\alpha}\sigma_{\mathrm{A}}^{\alpha}[\omega^{\alpha}(\beta_2^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_5^{\alpha})+\rho^{\alpha}(\eta_3^{\alpha}\eta_4^{\alpha}-\eta_1^{\alpha}\eta_6^{\alpha})]}{(\mu_{\mathrm{H}}^{\alpha}+\sigma_{\mathrm{H}}^{\alpha}+\kappa^{\alpha})(\mu_{\mathrm{H}}^{\alpha}+\gamma_{\mathrm{H}}^{\alpha})(\mu_{\mathrm{A}}+\sigma_{\mathrm{A}})(\mu_{\mathrm{A}}^{\alpha}+\gamma_{\mathrm{A}}^{\alpha})\omega^{\alpha}}>1. \end{aligned} \tag{63}$$

Eigenvalues have a negative real portion as a result of the trace and determinant criteria being proven. For every $\alpha\in]0,1]$, $|\arg\lambda_j|>\alpha\pi/2$. □

When $R_0<1$ and condition (63) holds, the DFE ψ_0 of the model (5) is locally asymptotically stable; otherwise, it is unstable.

3.6. Global Stability of DFE. Theorem by [19] is used to study the global asymptotic stability (GAS) of the model's DFE state. Based on (5),

$$\begin{cases} \dfrac{dV}{dt}=F(V,T), \\ \dfrac{dT}{dt}=G(V,T), \text{ with } G(V,0)=0. \end{cases} \tag{64}$$

The uninfected population consists of $V=(S_{\mathrm{H}},V_{\mathrm{H}},S_{\mathrm{A}})$, and infected population consists of $T=(E_{\mathrm{H}},I_{\mathrm{H}},E_{\mathrm{A}},I_{\mathrm{A}},C_{\mathrm{e}})$.

For the system $dV/dt=F(V,0)$, assume that V^* is DFE, with

$$V^*=\left(\frac{\Lambda_{\mathrm{H}}^{\alpha}(\phi^{\alpha}+(1-\kappa^{\alpha})\mu_{\mathrm{H}}^{\alpha})}{\mu_{\mathrm{H}}(\mu_{\mathrm{H}}^{\alpha}+\phi^{\alpha})},\frac{\kappa^{\alpha}\Lambda_{\mathrm{H}}^{\alpha}}{\mu_{\mathrm{H}}^{\alpha}+\phi^{\alpha}},\frac{\Lambda_{\mathrm{A}}^{\alpha}}{\mu_{\mathrm{A}}^{\alpha}}\right). \tag{65}$$

It is assured that the model's disease-free equilibrium (DFE) point Φ_0 is GAS if $R_0<1$, which is LAS (local asymptotical and stable).

(i) A1: for $dV/dt=F(V,0)$, V^* is GAS for model (5)

(ii) A2: $G(V,T)=AT-G^*(V,T)$, $G^*(V,T)\geq 0,\forall(V,T)\in\Psi$

The model is biologically meaningful if Ψ_0, and $A=(\partial G(\Phi_0))/\partial T$ is an M-matrix with nonnegative nondiagonal elements.

If the two aforementioned assumptions are met by model (5), then the following theorem holds.

Theorem 6. *If $R_0<1$ is LAS and assumptions A1 and A2 are true, then DFE, Φ_0, is GAS for model (5).*

Proof. In order to demonstrate that $V\longrightarrow V^*$, we must first demonstrate that assumptions A1 and A2 hold for $R_0<1$.

$$\begin{cases} {}_0^{C}D_t^{\alpha}S_{\mathrm{H}}(t)=\Lambda_{\mathrm{H}}^{\alpha}+\phi^{\alpha}V_{\mathrm{H}}-\kappa^{\alpha}\Lambda_{\mathrm{H}}^{\alpha}-\mu_{\mathrm{H}}^{\alpha}S_{\mathrm{H}}, \\ {}_0^{C}D_t^{\alpha}V_{\mathrm{H}}(t)=\kappa^{\alpha}\Lambda_{\mathrm{H}}^{\alpha}-(\mu_{\mathrm{H}}^{\alpha}+\phi^{\alpha})V_{\mathrm{H}}, \\ {}_0^{C}D_t^{\alpha}S_{\mathrm{A}}(t)=\Lambda_{\mathrm{A}}^{\alpha}-\mu_{\mathrm{A}}^{\alpha}S_{\mathrm{A}}. \end{cases} \tag{66}$$

The α's order linear ODEs are represented by the second and third equations of (66), and their solutions look like this ${}_0^{C}D_t^{\alpha}S_{\mathrm{H}}(t)=\Lambda_{\mathrm{H}}^{\alpha}+\phi^{\alpha}V_{\mathrm{H}}-\kappa^{\alpha}\Lambda_{\mathrm{H}}^{\alpha}-\mu_{\mathrm{H}}^{\alpha}S_{\mathrm{H}}$; using Laplace transform, we will get

$$(W^{\alpha}+\mu_{\mathrm{A}}^{\alpha})S_{\mathrm{A}}(W)=\frac{\Lambda_{\mathrm{A}}^{\alpha}}{W}\Rightarrow. \tag{67}$$

We can now obtain the following using the Mittag-Leffler function and the Laplace inverse transform of $S_A(W)$:

$$S_A(t) = \frac{\Lambda_A^\alpha}{\mu_A^\alpha}[1 - E_\alpha(-\mu_A^\alpha t^\alpha)] \text{ with } \mu_A^\alpha > 0. \tag{68}$$

Then, $S_A(t) \longrightarrow \Lambda_A^\alpha/\mu_A^\alpha$ if $t \longrightarrow \infty$.

Using the same technique, we get

$$V_H(t) = \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha)t^\alpha)] \text{ with } (\mu_A^\alpha + \phi^\alpha) > 0. \tag{69}$$

Then, $V_H(t) \longrightarrow \kappa^\alpha \Lambda_H^\alpha/\mu_H^\alpha + \phi^\alpha$ if $t \longrightarrow \infty$.

Subtracting $V_H(t)$ from the initial equation of (66) produces

$$D_t^\alpha S_H(t) = \Lambda_H^\alpha(1-\kappa^\alpha) - \mu_H^\alpha S_H + \phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}[1 - E_\alpha(-(\mu_H^\alpha + \phi^\alpha)t^\alpha)]. \tag{70}$$

We get the following from the Laplace transform of (70):

$$\begin{aligned} S_H(W) = {} & \frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{W(W^\alpha + \mu_H^\alpha)} + \phi^\alpha \frac{\kappa^\alpha \Lambda_H^\alpha}{(\mu_H^\alpha + \phi^\alpha)(W^\alpha + \mu_H^\alpha)W} \\ & - \frac{\phi^\alpha \kappa^\alpha \Lambda_H^\alpha}{(\mu_H^\alpha + \phi^\alpha)} \frac{1}{W^\alpha + \mu_H^\alpha} \times \frac{W^{\alpha-1}}{W^\alpha + (\mu_H^\alpha + \phi^\alpha)}. \end{aligned} \tag{71}$$

From the Laplace inverse transform, we obtain

$$\begin{aligned} S_H(t) = {} & \frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{\mu_H^\alpha}[1 - E_\alpha(-\mu_H^\alpha t^\alpha)] \\ & + \frac{\phi^\alpha \kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha(\mu_H^\alpha + \phi^\alpha)}[1 - E_\alpha(-\mu_H^\alpha t^\alpha)] - \frac{\phi^\alpha \kappa^\alpha \Lambda_H^\alpha}{(\mu_H^\alpha + \phi^\alpha)} \\ & \times t^{\alpha-1} E_{\alpha,\alpha}(-\mu_H^\alpha t^\alpha) \times E_{\alpha,1}[-(\mu_H^\alpha + \phi^\alpha)], \\ \lim_{t\longrightarrow\infty} S_H(t) = {} & \frac{\Lambda_H^\alpha(1-\kappa^\alpha)}{\mu_H^\alpha} + \frac{\phi^\alpha \kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha(\mu_H^\alpha + \phi^\alpha)} \\ = {} & \frac{\Lambda_H^\alpha[\phi^\alpha + \mu_H^\alpha(1-\kappa^\alpha)]}{\mu_H^\alpha(\mu_H^\alpha + \phi^\alpha)}. \end{aligned} \tag{72}$$

So all points concerning these conditions converge at

$$V^* = \left(\frac{\Lambda_H^\alpha(\phi^\alpha + (1-\kappa^\alpha)\mu_H^\alpha)}{\mu_H(\mu_H^\alpha + \phi^\alpha)}, \frac{\kappa^\alpha \Lambda_H^\alpha}{\mu_H^\alpha + \phi^\alpha}, \frac{\Lambda_A^\alpha}{\mu_A^\alpha}\right). \tag{73}$$

Thus, V^* is asymptotically stable globally.

Consider

$$G(V,T) = \begin{cases} G1(V,T) = \left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) S_H + d\left(\frac{\eta_1^\alpha I_H + \eta_2^\alpha I_A + \eta_3^\alpha C_e}{\Omega_H}\right) V_H - (\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) E_H, \\ G2(V,T) = \sigma_H^\alpha E_H - (\mu_H^\alpha + \gamma_H^\alpha) I_H, \\ G3(V,T) = \left(\frac{\eta_4^\alpha I_H + \eta_5^\alpha I_A + \eta_6^\alpha C_e}{\Omega_A}\right) S_A - (\mu_A^\alpha + \sigma_A^\alpha) E_A, \\ G4(V,T) = \sigma_A^\alpha E_A - (\mu_A^\alpha + \gamma_A^\alpha) I_A, \\ G5(V,T) = \rho^\alpha I_A - \omega^\alpha C_e. \end{cases} \tag{74}$$

We then obtain

$$\frac{\partial G}{\partial T} = \begin{bmatrix} -(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) & \frac{\eta_1^\alpha}{\Omega_H} S_H + d\frac{\eta_1^\alpha}{\Omega_H} V_H & 0 & \frac{\eta_2^\alpha}{\Omega_H} S_H + d\frac{\eta_2^\alpha}{\Omega_H} V_H & \frac{\eta_3^\alpha}{\Omega_H} S_H + d\frac{\eta_3^\alpha}{\Omega_H} V_H \\ \sigma_H^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 & 0 & 0 \\ 0 & \frac{\eta_4^\alpha}{\Omega_A} S_A & -(\mu_A^\alpha + \sigma_A^\alpha) & \frac{\eta_5^\alpha}{\Omega_A} S_A & \frac{\eta_6^\alpha}{\Omega_A} S_A \\ 0 & 0 & \sigma_A^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 \\ 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix}, \tag{75}$$

$$A = \frac{\partial G(V^*, 0)}{\partial T} = \begin{bmatrix} -(\mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha) & Y_1 & 0 & Y_2 & Y_3 \\ \sigma_H^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 & 0 & 0 \\ 0 & \eta_4^\alpha & -(\mu_A^\alpha + \sigma_A^\alpha) & \eta_5^\alpha & \eta_6^\alpha \\ 0 & 0 & \sigma_A^\alpha & -(\mu_H^\alpha + \gamma_H^\alpha) & 0 \\ 0 & 0 & 0 & \rho^\alpha & -\omega^\alpha \end{bmatrix},$$

where

$$Y_1 = \frac{\eta_1^\alpha \phi^\alpha + \eta_1^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha},$$
$$Y_2 = \frac{\eta_2^\alpha \phi^\alpha + \eta_2^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha},$$
$$Y_3 = \frac{\eta_3^\alpha \phi^\alpha + \eta_3^\alpha \mu_H^\alpha (1 - \kappa^\alpha + d\kappa^\alpha)}{\phi^\alpha + \mu_H^\alpha},$$
$$G^*(V,T) = AT - G(V,T) = \begin{bmatrix} (\eta_1^\alpha + \eta_2^\alpha + \eta_3^\alpha) I_H \left(1 - \frac{S_H + dV_H}{\Omega_H} + \mu_H^\alpha \kappa^\alpha (d-1)\right) \\ 0 \\ (\eta_4^\alpha + \eta_5^\alpha + \eta_6^\alpha) I_H \left(1 - \frac{S_A}{\Omega_A}\right) \\ 0 \\ 0 \end{bmatrix}. \tag{76}$$

We have $(S_H + dV_H)/\Omega_H \ll 1$ and $\mu_H^\alpha \kappa^\alpha (d-1) \ll 1$ since all parameters are positive as well. Since $G1 \geq 0$, $G3 \geq 0$ must also follow. □

As a result, $G^*(V,T) \geq 0 \forall (V,T) \in \Psi$.

Hence, model (5)'s DFE point Φ_0 is GAS.

3.7. bTB Endemic Equilibrium Points. Let $(S_H, E_H, V_H, I_H, S_A, E_A, I_A, C_e) \in \mathbb{R}_+^8$. $E^* = (S_H^*, E_H^*, V_H^*, I_H^*, S_A^*, E_A^*, I_A^*, C_e^*)$ denotes bTB endemic equilibrium point.

Given that

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

$$\begin{cases} S_H^* = \frac{[\Lambda_H (1 - \kappa^\alpha) + \phi^\alpha V_H^*] \Omega_H}{\mu_H^\alpha \Omega_H + (\eta_1 I_H^* + (\eta_2^\alpha + \eta_3^\alpha \rho^\alpha / \omega^\alpha) I_A^*)}, \\ E_H^* = \frac{\mu_H^\alpha + \gamma_H^\alpha}{\sigma_H \alpha} I_H^*, \\ V_H^* = \frac{\kappa^\alpha \Omega_H (\Lambda_H^\alpha \sigma_H^\alpha + \mu_H^\alpha + \gamma_H^\alpha)}{\Omega_H (\mu_H^\alpha + \phi^\alpha) + d(\eta_1 I_H^* + (\eta_2^\alpha + \eta_3^\alpha \rho^\alpha / \omega^\alpha) I_A^*)}, \\ I_H^* = \frac{(\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)[\Omega_A \mu_A^\alpha + (\eta_5^\alpha + \eta_6^\alpha \rho^\alpha / \omega^\alpha) I_A^*] - \Lambda_A^\alpha \sigma^\alpha (\eta_5^\alpha + \eta_5^\alpha \rho^\alpha / \omega_A^\alpha)}{\eta_4^\alpha \Lambda_H^\alpha \sigma_H^\alpha - \eta_4^\alpha (\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)}, \\ S_A^* = \frac{\Lambda_A}{\mu_A} - \frac{(\mu_A^\alpha + \gamma_A^\alpha)(\mu_A^\alpha + \sigma_A^\alpha)}{\mu_A^\alpha \sigma_A^\alpha} I_A^*, \\ E_A^* = \frac{\mu_A^\alpha + \gamma_A^\alpha}{\sigma_A^\alpha} I_A^*, \\ I_A^* = I_A^*, \\ C_e^* = \frac{\rho^\alpha}{\omega^\alpha} I_A^*. \end{cases} \tag{77}$$

Table 2: R_0 sensitivity indexes.

Parameters	Indexes
μ_A	-0.08
μ_H	-0.17
η_1	0.03
η_4	0.14
η_5	0.42
η_6	0.26
σ_A	0.03
σ_H	0.12
κ	-0.13
α_A	-0.78
α_H	-0.09
ρ	0.26
ϕ	-0.06
d	0.01
ω	-0.40

3.8. Global Stability of EE Points. Let $E^* = (S_H^*, E_H^*, V_H^*, I_H^*, S_A^*, E_A^*, I_A^*, C_e^*)$ be the global stability.

Theorem 7. *Consider $\alpha \in (0, 1]$ and $R_0 > 1$. Then, in the interior of Ψ, endemic equilibruim of the fractional-order model (5) is globally stable.*

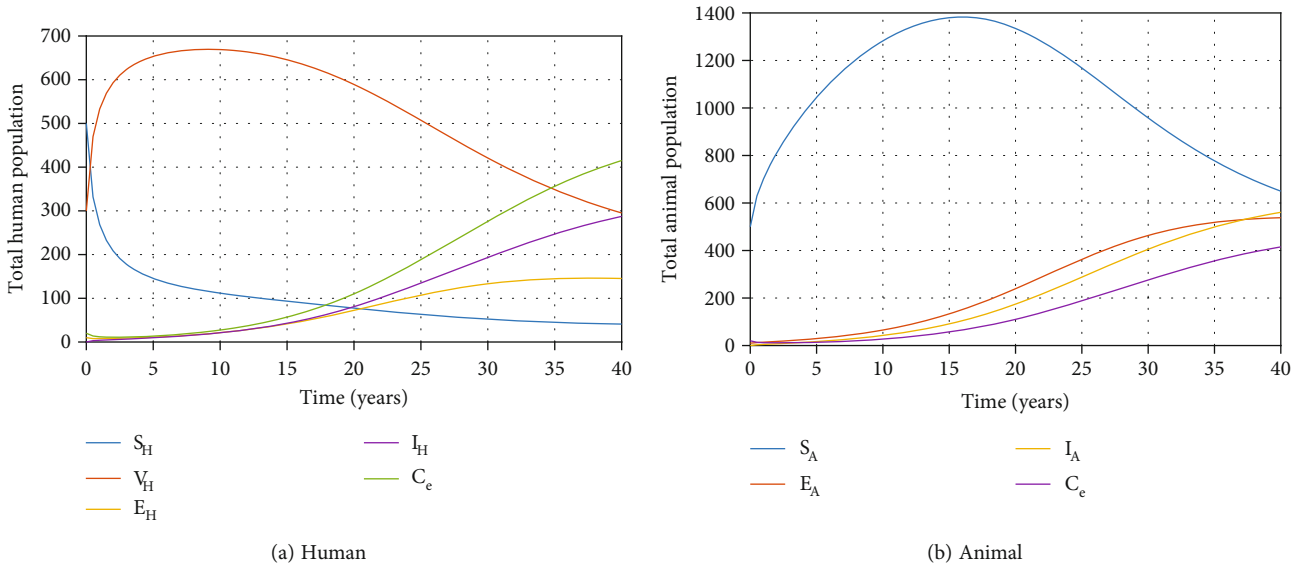
Proof. To define a function, we use the Volterra-type Lyapunov functional approach in [3, 20].

$L(t)$: $\varepsilon(t) = [S_H(t), E_H(t), V_H(t), I_H(t), S_A(t), E_A(t), I_A(t), C_e(t)]^T \longrightarrow \mathbb{R}$, as

$$\begin{aligned} L(t) = {} & \frac{1}{a_1}\left(S_H - S_H^* - S_H^* \log \frac{S_H}{S_H^*}\right) + \frac{1}{a_2}\left(E_H - E_H^* - E_H^* \log \frac{E_H}{E_H^*}\right) \\ & + \frac{1}{a_3}\left(V_H - V_H^* - V_H^* \log \frac{V_H}{V_H^*}\right) + \frac{1}{a_4}\left(I_H - I_H^* - I_H^* \log \frac{I_H}{I_H^*}\right) \\ & + \frac{1}{a_5}\left(S_A - S_A^* - S_A^* \log \frac{S_A}{S_A^*}\right) + \frac{1}{a_6}\left(E_A - E_A^* - E_A^* \log \frac{E_A}{E_A^*}\right) \\ & + \frac{1}{a_7}\left(I_A - I_A^* - I_A^* \log \frac{I_A}{I_A^*}\right) + \frac{1}{a_8}\left(C_e - C_e^* - C_e^* \log \frac{C_e}{C_e^*}\right), \end{aligned} \tag{78}$$

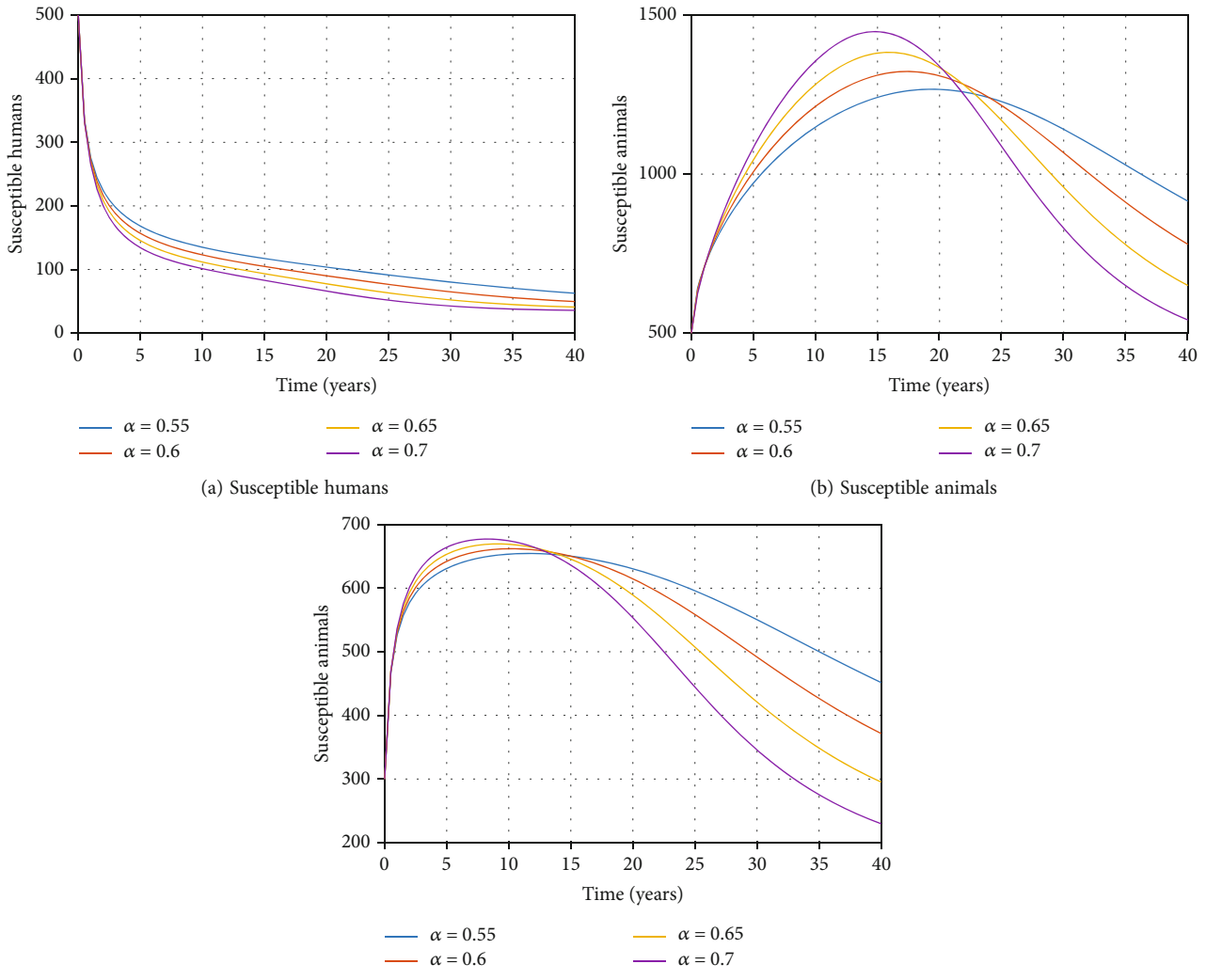
where

$$\begin{aligned} a_1 &= \lambda_H^\alpha + \mu_H^\alpha, \\ a_2 &= \mu_H^\alpha + \sigma_H^\alpha + \kappa^\alpha, \\ a_3 &= \mu_H^\alpha + \phi^\alpha + d\lambda_H^\alpha, \\ a_4 &= \mu_H^\alpha + \gamma_H^\alpha, \\ a_5 &= \lambda_A^\alpha + \mu_A^\alpha, \\ a_6 &= \mu_A^\alpha + \sigma_A^\alpha, \\ a_7 &= \mu_A^\alpha + \gamma_A^\alpha, \\ a_8 &= \omega^\alpha. \end{aligned} \tag{79}$$



(a) Human (b) Animal

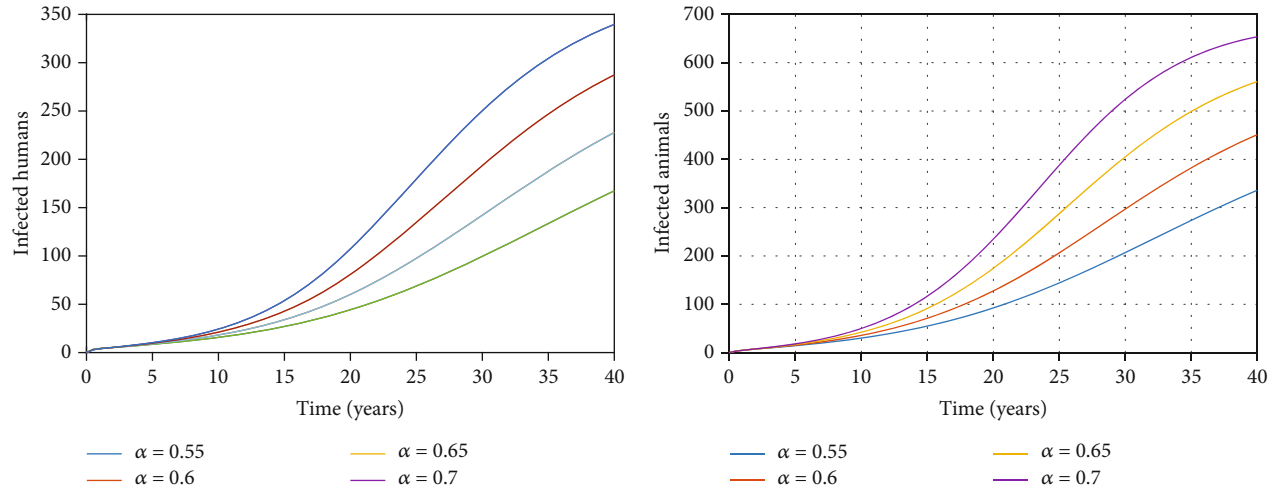
FIGURE 2: Bovine tuberculosis dynamics in the human and animal populations for $\alpha = 65$.



(a) Susceptible humans (b) Susceptible animals

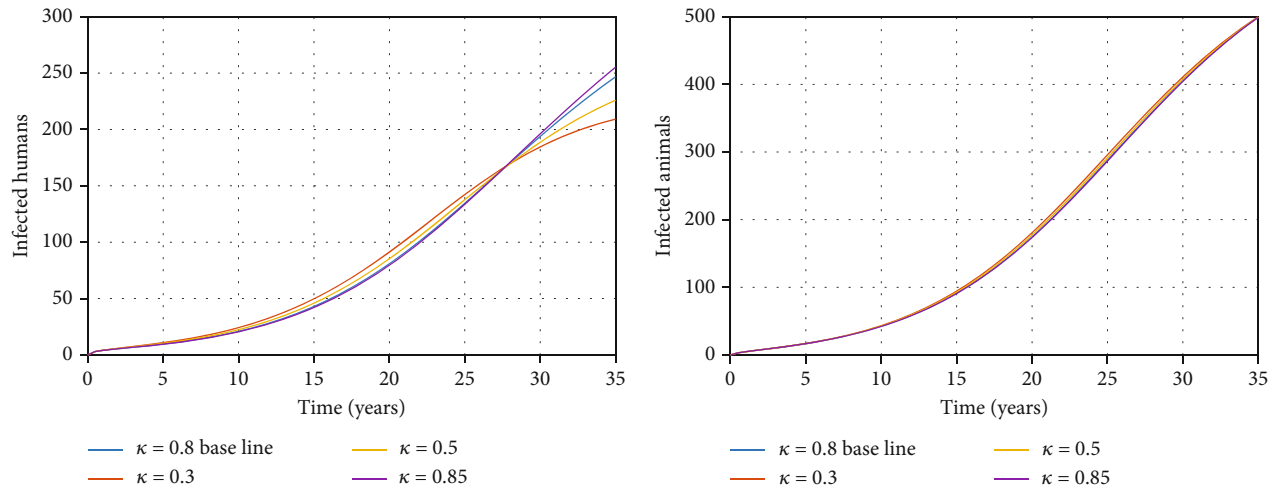
(c) Vaccinated humans

FIGURE 3: α variation for a given population.



(a) Infected humans (b) Infected animals

FIGURE 4: α variation for the infected population.



(a) Infected humans (b) Infected animals

FIGURE 5: Variation of κ.

$L(t)$ is specified as a continuous function and definite positive for any $t > 0$. 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 equivalence may be demonstrated [3].

Let us demonstrate that $D_t^\alpha L \le 0$ at the EE point.

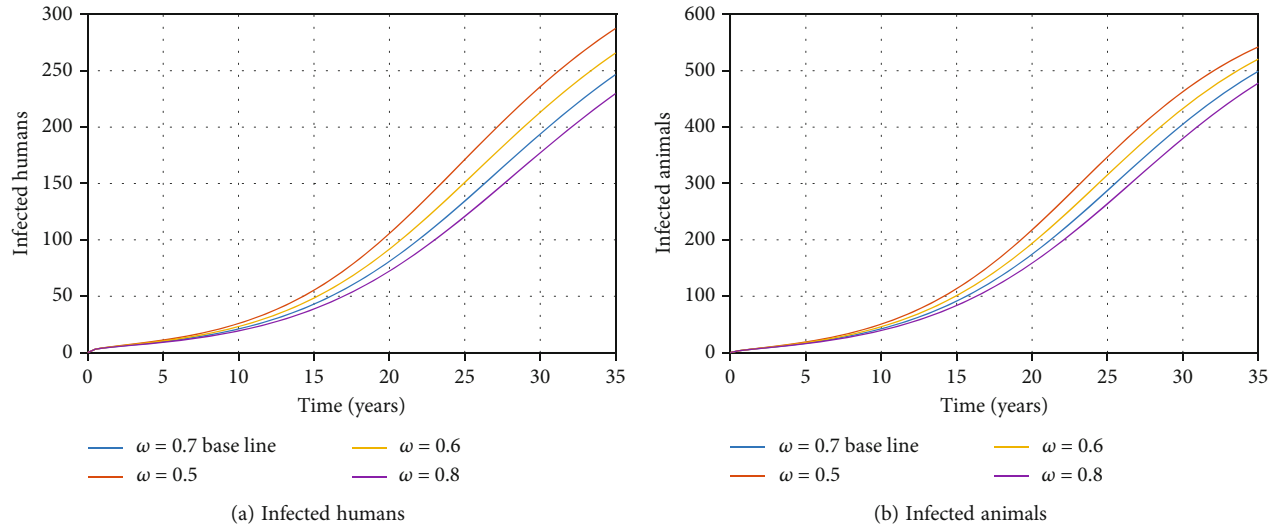
$$\begin{aligned} D_t^\alpha L = {} & \frac{1}{a_1}\left(\frac{S_H - S_H^*}{S_H}\right) D_t^\alpha S_H + \frac{1}{a_2}\left(\frac{E_H - E_H^*}{E_H}\right) D_t^\alpha E_H \\ & + \frac{1}{a_3}\left(\frac{V_H - V_H^*}{V_H}\right) D_t^\alpha V_H + \frac{1}{a_4}\left(\frac{I_H - I_H^*}{I_H}\right) D_t^\alpha I_H \\ & + \frac{1}{a_5}\left(\frac{S_A - S_A^*}{S_A}\right) D_t^\alpha S_A + \frac{1}{a_6}\left(\frac{E_A - E_A^*}{E_A}\right) D_t^\alpha E_A \\ & + \frac{1}{a_7}\left(\frac{I_A - I_A^*}{S_A}\right) D_t^\alpha I_A + \frac{1}{a_8}\left(\frac{C_e - C_e^*}{C_e}\right) D_t^\alpha C_e. \end{aligned} \tag{80}$$

We get from Equation (80) the following by simplification:

$$\begin{aligned} D_t^\alpha L = {} & -\frac{(S_H - S_H^*)^2}{S_H} - \frac{(E_H - E_H^*)^2}{E_H} - \frac{(V_H - V_H^*)^2}{V_H} \\ & - \frac{(I_H - I_H^*)^2}{I_H} - \frac{(S_A - S_A^*)^2}{S_A} - \frac{(E_A - E_A^*)^2}{E_A} \\ & - \frac{(I_A - I_A^*)^2}{S_A} - \frac{(C_e - C_e^*)^2}{C_e}. \end{aligned} \tag{81}$$

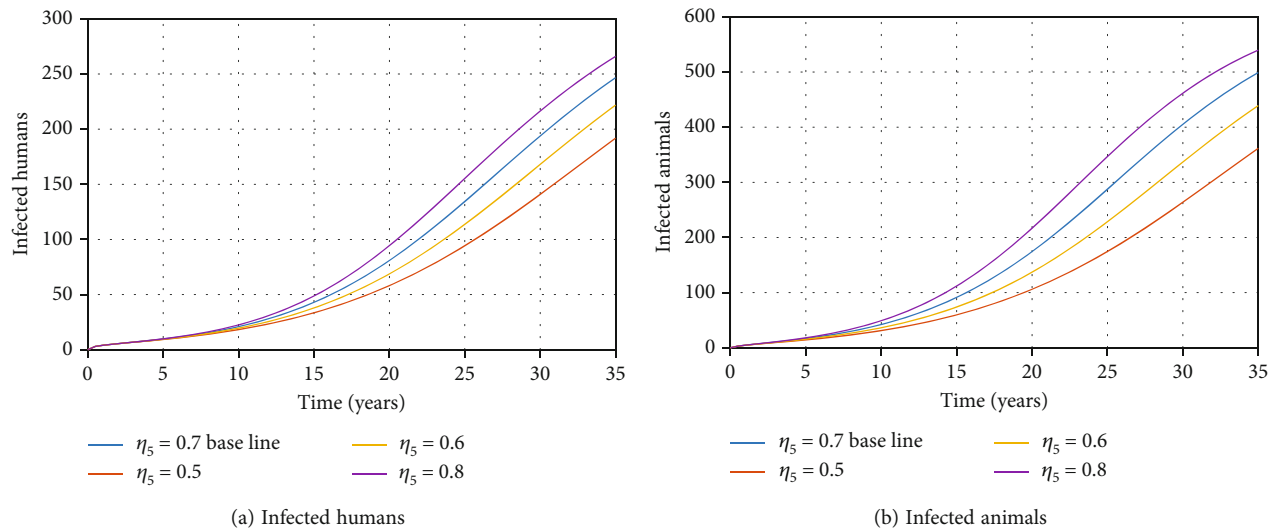
It can be established that $D_t^\alpha L \le 0$.

Hence, if $R_0 > 1$, (81) is less than zero (0) and equivalent to zero (0) if $S_H = S_H^*$, $E_H = E_H^*$, $V_H = V_H^*$, $I_H = I_H^*$, $S_A = S_A^*$, $E_A = E_A^*$, $I_A = I_A^*$, $C_e = C_e^*$.



(a) Infected humans (b) Infected animals

Figure 6: Variation of ω.



(a) Infected humans (b) Infected animals

Figure 7: Variation of η_5 for infected population.

Solutions in Ψ converge to E^* by LaSalle's invariance principle [3, 20, 21]. Consequently, if $R_0 > 1$, then (5) is GAS. □

4. Numerical Simulations

4.1. Reproductive Rate in the Absence of Immunization. Let R_0^* represent R_0 without vaccination. R_0^* and R_0 are obtained using parameter values in Table 1:

(i) Without vaccination: $R_0^* = 7.4298$

(ii) With vaccination $R_0 = 4.9575$

Increasing vaccination rates in human and animal populations is most effective in reducing bovine tuberculosis.

4.1.1. Threshold of Herd Immunity. Let H_1 denotes the Herd immunity; then,

$$H_1 = 1 - \frac{1}{R_0^*} = 0.86. \tag{82}$$

This indicates that 86% of humans and animals should receive vaccination if $R_0^* = 7.4298$.

4.2. Sensitivity Analysis. The sensitivity analysis of parameters in Table 2 of R_0 determines the contribution of each parameter [22]. Using the normalized forward sensitivity analysis in [22, 23],

$$\Psi_\beta^{R_0} = \left(\frac{\partial R_0}{\partial \beta}\right)\left(\frac{\beta}{R_0}\right). \tag{83}$$

For every 10% increase in dairy products, R_0 rises by 0.018%. There is an increase in the human disease-induced death rate γ_H, the human natural mortality rate μ_H, and the animal natural mortality rate α_A.

4.3. Numerical Simulation. We demonstrate the model behaviour by varying fractional orders $\alpha \in]0, 1]$ and parameter values. We use values in Table 1 for our simulations.

As shown in Figure 2, the number of susceptible individuals and animals decreases. However, susceptible class reduces more than animals, due to human vaccination.

The effects of changing α on humans, animals, and vaccinated humans are depicted in Figures 3(a)–3(c), respectively.

As seen in Figure 4, the population of animal appears more infected than in human population. This can be attributed or explained through vaccinations.

4.3.1. Influence of Vaccination Rate on Animals and Humans Who Are Infected. Figure 5 illustrates how the vaccination rate κ varies across the human population while maintaining constant values for other parameters. It can be observed that, as κ rises, the results have no impact on the animal population. The biological implication is that the number of infected individuals falls as the vaccination rate κ increases.

4.3.2. Impact of Decay Rate on the Environment That Is Contaminated. But while the other parameters remain constant, changing the rate of decay ω for the contaminated environment (meat and dairy products) demonstrates in Figure 6 a direct correlation between an increase in infectious humans and animals. A decrease in the decay rate plays a major role in eliminating the illness in both human and animal populations.

4.3.3. Effects of the Rate at Which Animals Become Infected. The outcome of adjusting the animal infection rate η_5 while holding other constant parameter is shown in Figure 7. A rise in η_5 from 0.5 to 0.8 results in a higher number of individuals and animals afflicted.

The percentage of ill individuals and animals is higher for $\eta_5 = 0.8$. Based on numerical findings, there is a direct correlation between the rate of animal infection and the quantity of humans and animals that become infected.

To reduce the animal infection rate η_5 from affected animals and halt the spread of the disease, policy makers and health authorities should think about strategies to isolate infectious animals.

5. Conclusion

We created a fractional-order mathematical framework that outlines the evolution of bovine tuberculosis, using environmental pollution and vaccination as compartments. In Section 3, we examined the model's qualitative behaviours by defining the area that is feasible, the solution's positivity, equilibrium points and their global and local stability, and the model's fundamental reproduction number.

The reproduction rate was subjected to a sensitivity analysis, which demonstrated a significant impact on the management of tuberculosis in cows. A parameter contribution analysis and numerical simulation were performed. The effect of parameters κ, ω, and eta_5 on the fractional-order model was explored.

Based on our findings, we can infer that a substantial reduction in the rate of bovine tuberculosis transmission in both human and animal populations can be achieved by raising the vaccination rate, κ, of both populations. Controlling the disease requires reducing the rate of animal infection from diseased animals eta_5 while raising the pace at which the contaminated habitat decays.

Data Availability

The data used to support the conclusion of the study are included in the paper.

Conflicts of Interest

The authors declare no conflict of interest.

Acknowledgments

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