



Nonlinear Dynamics of a Zoonotic Disease With Control Interventions Through Fractional Derivative

Rashid Jan^{1,2}, Salah Boulaaras^{3,*}, Asma Alharbi³, Normy Norfiza Abdul Razak¹

¹ *Institute of Energy Infrastructure (IEI), Department of Civil Engineering, College of Engineering, Universiti Tenaga Nasional (UNITEN), Putrajaya Campus, Jalan IKRAM-UNITEN, 43000 Kajang, Selangor, Malaysia*

² *Mathematics Research Center, Near East University TRNC, Mersin 10, Nicosia 99138, Turkey*

³ *Department of Mathematics, College of Science, Qassim University, Buraydah, 51452, Saudi Arabia*

Abstract. The zoonotic infection campylobacteriosis poses a significant public health burden, contributing to widespread morbidity and, in severe cases, mortality, particularly among vulnerable populations such as children, the elderly, and immunocompromised individuals. The economic impact is considerable, with costs arising from medical care, hospitalization, lost productivity, and the need for stringent food safety measures. In this paper, we model the dynamics of campylobacteriosis with drug resistance in humans and animals using fractional derivatives. The fundamental concepts of fractional derivatives are presented to analyze the disease dynamics. Our work focuses on both the quantitative and qualitative analysis of the proposed model. The fixed-point theorem is applied to investigate the existence and uniqueness of solutions. We also examine the stability of the system through analytical techniques. To further explore the system, a numerical scheme is introduced to visualize the solution pathways and assess the influence of various factors. We demonstrate the dynamics of campylobacteriosis with drug resistance, highlighting the effects of different factors on infection levels. Furthermore, our results identify the key factors crucial for effective disease control and management.

2020 Mathematics Subject Classifications: 92D25, 92D30

Key Words and Phrases: Fractional calculus, Epidemic model, Stability analysis, Fixed point theory, Disease control, Public health

1. Introduction

The most common bacterial infection, campylobacteriosis, which primarily causes gastroenteritis in humans, is mainly caused by *Campylobacter coli* and *Campylobacter jejuni*

*Corresponding author.

DOI: <https://doi.org/10.29020/nybg.ejpam.v17i4.5534>

Email addresses: rashid.ash2000@yahoo.com (R. Jan), s.boulaaras@qu.edu.sa (S. Boulaaras), ao.alharbi@qu.edu.sa (A. Alharbi), normy@uniten.edu.my (N. N. A. Razak)

[20]. The disease is primarily zoonotic, meaning it is transmitted from animals to humans, with the most common sources being contaminated poultry, untreated water, and raw milk. Symptoms include abdominal pain, fever, diarrhea, and nausea, typically lasting from several days to a week [5]. In severe cases, particularly in vulnerable populations such as young children, the elderly, and immunocompromised individuals, the infection can lead to complications such as bacteremia or neurological conditions like Guillain-Barré syndrome. The seasonal nature of campylobacteriosis, often peaking in warmer months, underscores the importance of continuous monitoring and food safety practices to reduce transmission rates [6]. The growing concern over antimicrobial resistance (AMR) in *Campylobacter* species poses a significant challenge for treatment. Resistance to commonly used antibiotics, such as fluoroquinolones and macrolides, has increased, making severe cases more difficult to manage and prolonging illness duration [9]. This rising resistance highlights the need for stricter regulatory measures in both human medicine and agriculture, where antibiotic use in livestock contributes to resistance. Addressing campylobacteriosis requires a multifaceted approach, including improved hygiene in food handling, water treatment, and surveillance to prevent outbreaks. Additionally, research aimed at understanding the dynamics of campylobacter transmission and resistance is crucial for developing effective public health interventions and safeguarding food safety [4].

Mathematical models are essential tools for representing real-world problems, and numerous techniques have been developed to effectively conceptualize these models [2]. Several mathematical models have been developed to study the transmission dynamics of campylobacteriosis. A comprehensive understanding of the dynamics of campylobacteriosis at both policy and implementation levels in public health is essential for devising optimal control strategies and cost-effective prevention measures [4]. Deterministic models are critical in enhancing this understanding by offering a theoretical framework that identifies key factors contributing to the spread and control of the disease [8, 13]. Deterministic modeling involves the development, testing, and validation of models that mathematically represent natural phenomena, systems, or hypotheses. These models provide a systematic approach to understanding and predicting the behavior of such phenomena from a mathematical perspective [18, 19]. The emergence of drug-resistant *Campylobacter* strains has become a major public health issue [24]. The overuse of antibiotics, especially in livestock, has fueled resistance, making infections harder to treat. Drug-resistant *Campylobacter* spreads between animals and humans through contaminated food, water, or direct contact, complicating control efforts [23]. This resistance significantly reduces the efficacy of antibiotics such as fluoroquinolones and macrolides, thereby increasing the risk of severe illness. Understanding the dynamics of drug-resistant campylobacteriosis is crucial for formulating effective strategies to mitigate its spread and safeguard public health. Consequently, this study investigates the transmission dynamics of campylobacteriosis in both human and animal populations, with a particular emphasis on the implications of drug resistance.

Fractional calculus offers several advantages for modeling real-world problems, particularly in fields like biology, engineering, finance, and physics [7, 22]. The application of fractional calculus to real-world problems facilitates a deeper understanding of complex

systems, leading to more effective solutions and interventions in various fields [3, 14]. The use of non-integer framework in modeling biological phenomena enhances the accuracy, flexibility, and comprehensiveness of mathematical representations, facilitating a better understanding of the complex dynamics inherent in biological systems [15, 21]. This effectiveness ultimately contributes to improved predictions, analyses, and interventions in various biological and public health contexts [1]. This flexibility of fractional calculus enhances the realism of mathematical models, facilitating the exploration of non-linear interactions and complex feedback mechanisms inherent in biological processes [11, 16]. Consequently, fractional calculus functions as a robust framework for enhancing our understanding of biological dynamics, thereby facilitating the development of more effective strategies for disease control, treatment planning, and public health interventions. Thus, we have opted to model the dynamics of campylobacteriosis using fractional calculus to achieve more detailed and reliable results for best control policies.

The research is structured as follows: Section 2 outlines the core ideas and principles of fractional theory. Section 3 introduces a model designed to capture the transmission dynamics of campylobacteriosis in humans and animals with drug resistance. In Section 4, a thorough analysis of the campylobacteriosis dynamics is conducted. Necessary stability conditions are established in Section 5. In addition to this, a computational technique is presented to illustrate the system's solutions under varying input factors. The final section provides a summary and concludes with final remarks.

2. Theory and results

Here, we present the ideas of Caputo's fractional operator to investigate the dynamics of campylobacteriosis. The fundamental concepts and theory are detailed as:

Definition 1. [17]. Suppose $b(t)$ such that $b(t) \in L^1([g, h], R)$, then the integral Caputo is

$${}^h I_{g^+}^{\bar{h}} b(t) = \frac{1}{\Gamma(\bar{h})} \int_0^t (t-r)^{\bar{h}-1} b(r) dr, \quad (1)$$

where \bar{h} is the fractional order with $0 < \bar{h} \leq 1$.

Definition 2. [17]. If we take $b(t)$ such that $b(t) \in C^n[g, h]$, then the Caputo derivative is presented as

$${}^{LC} D_{0^+}^{\bar{h}} b(t) = \frac{1}{\Gamma(n-\bar{h})} \int_0^t (t-r)^{n-\bar{h}-1} b^{(n)}(r) dr. \quad (2)$$

Lemma 1. [17]. Suppose $b(h)$ and take the subsequent system

$$\begin{cases} {}^{LC} D_{0^+}^{\bar{h}} b(t) = u(t), t \in [0, \tau], \\ b(0) = u_0, \quad n-1 < \bar{h} < n, \end{cases} \quad (3)$$

whereas $u(t)$ belongs to $C([0, \tau])$ and

$$b(t) = \sum_{i=0}^{n-1} d_i t^i, \text{ for } i = 0, 1, \dots, n-1 \text{ and } d_i \in R.$$

Definition 3. For Caputo operator, the Laplace transformation is expressed as

$$\mathcal{L}[{}^{LC}D_{0+}^{\hbar}b(t)] = r^{\hbar}b(r) - \sum_{k=0}^{n-1} r^{\hbar-k-1}b^{(k)}(0), \quad (4)$$

whereas $n - 1 < \hbar < n$. Additionally, the norm on \mathcal{X} is defined as

$$\|b\| = \max_{t \in [0, \tau]} \{|b|\}, \text{ for all } b \in \mathcal{X}. \quad (5)$$

Theorem 1. [10]. Suppose \mathcal{X} denote a Banach space, with the condition that the mapping $G : \mathcal{X} \rightarrow \mathcal{X}$ is simultaneously compact and continuous. If

$$\mathcal{E} = \{b \in \mathcal{X} : b = \lambda Gb, \lambda \in (0, 1)\}, \quad (6)$$

is bounded, then, there is a fixed point of G .

3. Formulation of fractional dynamics

Here, we formulate the transmission dynamics of campylobacteriosis in animals and humans with drug resistance. The animal population is denoted by \mathcal{N}_a , while the human population is symbolized by \mathcal{N}_h . In this formulation, each population is further distributed into three classes: susceptible, infected, and recovered individuals. We assumed the recruitment and natural death rate of humans by Ξ_h and μ_h while the recruitment and natural death of the animals is denoted by Ξ_a and μ_a .

Regarding the population of infected humans, they undergo treatment at a consistent rate of $\epsilon_1\theta_1$, where ϵ_1 denotes the efficacy of the drug, and the recovery due to drug is indicated by τ_1 . In addition to this, $\epsilon_1\tau_1p_1\mathcal{I}_h$ shows the strength of \mathcal{I}_h who show resistance to the drug, with p_1 ranging between 0 and 1 signifying the ratio of resistance acquisition to the drug. Therefore, the expression $\epsilon_1\tau_1(1 - p_1)\mathcal{I}_h$ signifies the fraction of susceptible individuals affected by the drug. Moreover, a segment of the \mathcal{I}_h undergoes spontaneous recovery at a rate denoted by δ , while another portion succumbs to the infection at a rate of ρ . In the same way for animals population, they undergo treatment at a consistent rate of $\epsilon_2\theta_2$, where ϵ_2 denotes the efficacy of the drug, and the recovery due to drug is indicated by τ_2 . In addition to this, $\epsilon_2\tau_2p_2\mathcal{I}_a$ show strength of \mathcal{I}_a who show resistance to the drug, with p_2 ranging between 0 and 1 signifying the ratio of resistance acquisition to the drug. Therefore, the expression $\epsilon_2\tau_2(1 - p_2)\mathcal{I}_a$ signifies the fraction of susceptible animals affected by the drug. Moreover, a segment of the \mathcal{I}_a undergoes spontaneous recovery at a rate denoted by δ , while another portion succumbs to the infection at a rate of ρ . In Figure 1, the flow chart of the infection has been illustrated to highlight the overall phenomena. Then, we have the dynamics of campylobacteriosis in term of ODE

with the above assumptions is given by

$$\begin{cases} \frac{d\mathcal{I}_h}{dt} = \Xi_h - \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_h + \nu_h\mathcal{R}_h - \mu_h\mathcal{I}_h, \\ \frac{d\mathcal{S}_h}{dt} = \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_h - \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h - (\gamma_h + \alpha_h + \mu_h)\mathcal{I}_h, \\ \frac{d\mathcal{R}_h}{dt} = \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h + \gamma_h\mathcal{I}_h - (\nu_h + \mu_h)\mathcal{R}_h, \\ \frac{d\mathcal{I}_a}{dt} = \Xi_a - \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_a + \nu_a\mathcal{R}_a - \mu_a\mathcal{I}_a, \\ \frac{d\mathcal{S}_a}{dt} = \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_a - \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a - (\gamma_a + \alpha_a + \mu_a)\mathcal{I}_a, \\ \frac{d\mathcal{R}_a}{dt} = \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a + \gamma_a\mathcal{I}_a - (\nu_a + \mu_a)\mathcal{R}_a, \end{cases} \tag{7}$$

where

$$0 \leq \mathcal{I}_h(0), 0 \leq \mathcal{S}_h(0), 0 \leq \mathcal{R}_h(0), 0 \leq \mathcal{I}_a(0), 0 \leq \mathcal{S}_a(0) \text{ and } 0 \leq \mathcal{R}_a(0).$$

Additionally, the size of animals

$$\mathcal{N}_a = \mathcal{I}_a + \mathcal{S}_a + \mathcal{R}_a,$$

similarly, the size of humans

$$\mathcal{N}_h = \mathcal{I}_h + \mathcal{S}_h + \mathcal{R}_h.$$

A fractional framework for an epidemic model involves incorporating fractional-order

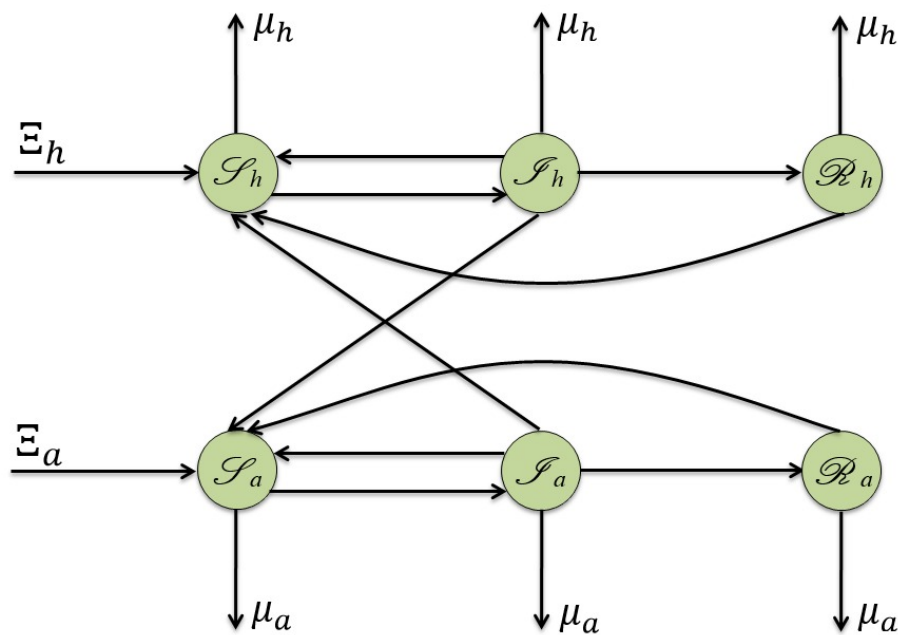


Figure 1: Flow chart of the dynamics of campylobacteriosis.

differential equations or fractional calculus principles into the traditional models used to describe the spread of infectious diseases. By introducing fractional derivatives or integrals, the model can capture memory effects and long-range interactions, providing a

more accurate description of certain epidemiological phenomena. Therefore, we represent the above model of malaria via non-integer derivative as

$$\begin{cases} {}^LC D_t^\vartheta \mathcal{S}_h &= \Xi_h - \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{S}_h + \nu_h\mathcal{R}_h - \mu_h\mathcal{S}_h, \\ {}^LC D_t^\vartheta \mathcal{I}_h &= \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{S}_h - \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h - (\gamma_h + \alpha_h + \mu_h)\mathcal{I}_h, \\ {}^LC D_t^\vartheta \mathcal{R}_h &= \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h + \gamma_h\mathcal{I}_h - (\nu_h + \mu_h)\mathcal{R}_h, \\ {}^LC D_t^\vartheta \mathcal{S}_a &= \Xi - \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{S}_a + \nu_a\mathcal{R}_a - \mu_a\mathcal{S}_a, \\ {}^LC D_t^\vartheta \mathcal{I}_a &= \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{S}_a - \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a - (\gamma_a + \alpha_a + \mu_a)\mathcal{I}_a, \\ {}^LC D_t^\vartheta \mathcal{R}_a &= \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a + \gamma_a\mathcal{I}_a - (\nu_a + \mu_a)\mathcal{R}_a. \end{cases} \tag{8}$$

In the above, the Liouville-Caputo’s operator is indicated by ${}^LC D_t^\vartheta$, with the index of memory represented by the symbol ϑ . The results derived from fractional systems offer increased reliability and precision owing to the non-local characteristics inherent in biological processes. Additionally, fractional systems possess a hereditary property, offering insights into both past and current states for future predictions. Recognizing Caputo’s derivative as a more dependable and versatile analytical tool, we have depicted the dynamics of campylobacteriosis infection within the framework of fractional calculus.

Theorem 2. *The solutions $(\mathcal{S}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a, \mathcal{R}_a)$ to the fractional system (8) describing campylobacteriosis are both non-negative and bounded.*

Proof. To prove the theorem, we proceed as

$$\begin{cases} {}^LC D_t^\vartheta \mathcal{S}_h |_{\mathcal{S}_h=0} &= \Xi_h + \nu_h\mathcal{R}_h \geq 0, \\ {}^LC D_t^\vartheta \mathcal{I}_h |_{\mathcal{I}_h=0} &= \beta_h\mathcal{S}_h\mathcal{I}_h \geq 0, \\ {}^LC D_t^\vartheta \mathcal{R}_h |_{\mathcal{R}_h=0} &= \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h + \gamma_h\mathcal{I}_h \geq 0, \\ {}^LC D_t^\vartheta \mathcal{S}_a |_{\mathcal{S}_a=0} &= \Xi_a + \nu_a\mathcal{R}_a \geq 0, \\ {}^LC D_t^\vartheta \mathcal{I}_a |_{\mathcal{I}_a=0} &= \beta_a\mathcal{S}_h\mathcal{I}_a \geq 0, \\ {}^LC D_t^\vartheta \mathcal{R}_a |_{\mathcal{R}_a=0} &= \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a + \gamma_a\mathcal{I}_a \geq 0. \end{cases} \tag{9}$$

Thus, the solutions of (8) are non-negative. For the boundedness of solution, we initially sum all compartments within the host population.

$${}^LC D_t^\vartheta (\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h) \leq \Xi_h - \mu_h(\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h), \tag{10}$$

this implies that

$$\left((\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h) \right) \leq \left(\mathcal{S}_h(0) + \mathcal{I}_h(0) + \mathcal{R}_h(0) - \frac{\Xi_h}{\mu_h} \right) E_\vartheta(-\mu_h t^\vartheta) + \frac{\Xi_h}{\mu_h}.$$

The obtained result is derived the theory of the work [17], we have the following

$$\left(\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h \right) \leq \frac{\Xi_h}{\mu_h} \cong \mathcal{M}_1.$$

In the same way, taking the compartments of animal population of the system (8), we find that $\mathcal{S}_a + \mathcal{I}_a + \mathcal{R}_a \leq \mathcal{M}_2$, where $\mathcal{M}_2 = \frac{\Xi_a}{\mu_a}$. Consequently, the solutions of the campylobacteriosis dynamics (8) exhibit both positivity and boundedness.

Now, denote the disease-free steady state of (8) by $\mathcal{E}_0(\mathcal{I}_h^0, \mathcal{I}_h^0, \mathcal{R}_h^0, \mathcal{I}_a^0, \mathcal{I}_a^0, \mathcal{R}_a^0)$ which is

$$\left(\frac{\Xi_h}{\mu_h}, 0, 0, \frac{\Xi_a}{\mu_a}, 0 \right).$$

4. Existence theory

The existence theory of epidemic models plays a vital role in validating and understanding the dynamics of infectious diseases, guiding public health strategies, and facilitating the development of numerical methods for analysis and prediction. This theoretical framework is essential for effectively addressing and managing epidemics in a scientifically sound manner. Here, we will analyze the qualitative aspects of the dynamics (8) of campylobacteriosis through existence theory. To achieve this, take the following steps:

$$\begin{cases} \mathcal{V}_1(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \Xi_h - \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_h + \nu_h\mathcal{R}_h - \mu_h\mathcal{I}_h, \\ \mathcal{V}_2(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \beta_h(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_h - \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h - (\gamma_h + \alpha_h + \mu_h)\mathcal{I}_h, \\ \mathcal{V}_3(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \epsilon_1\tau_1(1 - p_1)\mathcal{I}_h + \gamma_h\mathcal{I}_h - (\nu_h + \mu_h)\mathcal{R}_h, \\ \mathcal{V}_4(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \Xi_a - \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_a + \nu_a\mathcal{R}_a - \mu_a\mathcal{I}_a, \\ \mathcal{V}_5(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \beta_a(\mathcal{I}_h + \mathcal{I}_a)\mathcal{I}_a - \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a - (\gamma_a + \alpha_a + \mu_a)\mathcal{I}_a, \\ \mathcal{V}_6(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a) &= \epsilon_2\tau_2(1 - p_2)\mathcal{I}_a + \gamma_a\mathcal{I}_a - (\nu_a + \mu_a)\mathcal{R}_a. \end{cases} \tag{11}$$

Here, we can generalized the system (11) as follows:

$$\begin{cases} {}^{LC}D_{0+}^\vartheta \mathcal{Y}(t) &= \mathcal{L}(t, \mathcal{Y}(t)), \quad t \in [0, \tau], \\ \mathcal{Y}(0) &= \mathcal{Y}_0, \quad 0 < \vartheta \leq 1, \end{cases} \tag{12}$$

with

$$\begin{cases} \mathcal{Y}(t) = \begin{pmatrix} \mathcal{I}_h(t), \\ \mathcal{I}_h(t), \\ \mathcal{R}_h(t), \\ \mathcal{I}_a(t), \\ \mathcal{I}_a(t), \\ \mathcal{R}_a(t). \end{pmatrix}, \quad \mathcal{Y}_0(t) = \begin{pmatrix} \mathcal{I}_{h0}, \\ \mathcal{I}_{h0}, \\ \mathcal{R}_{h0}, \\ \mathcal{I}_{a0}, \\ \mathcal{I}_{a0}, \\ \mathcal{R}_{a0}. \end{pmatrix}, \quad \mathcal{L}(t, \mathcal{Y}(t)) = \begin{pmatrix} \mathcal{Y}_1(t, \mathcal{I}_{h1}, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_v, \mathcal{I}_v), \\ \mathcal{Y}_2(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a), \\ \mathcal{Y}_3(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a), \\ \mathcal{Y}_4(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a), \\ \mathcal{Y}_5(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a), \\ \mathcal{Y}_6(t, \mathcal{I}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{I}_a, \mathcal{I}_a, \mathcal{R}_a). \end{pmatrix} \end{cases} \tag{13}$$

By utilizing the aforementioned Lemma (1), we have the opportunity to represent the system (12) in a corresponding integral formulation, as presented below:

$$\mathcal{Y}(t) = \mathcal{Y}_0(t) + \frac{1}{\Gamma(\vartheta)} \int_0^t (t - r)^{\vartheta-1} \mathcal{L}(r, \mathcal{Y}(r)) dr. \tag{14}$$

To assess further, we utilized the following criteria based on Lipschitz conditions:

(C1) For q in the interval $[0, 1)$, there are corresponding sets $\mathcal{U}_{\mathcal{Y}}$ and $\mathcal{V}_{\mathcal{Y}}$ such that the below fulfills:

$$|\mathcal{L}(t, \mathcal{Y}(t))| \leq \mathcal{U}_{\mathcal{Y}} |\mathcal{Y}|^q + \mathcal{V}_{\mathcal{Y}}. \tag{15}$$

(C2) One can find $M_{\mathcal{L}} > 0$, and for all \mathcal{Y} and $\bar{\mathcal{Y}}$ belonging to the set \mathcal{X} , subject to the condition

$$|\mathcal{L}(t, \mathcal{Y}) - \mathcal{L}(t, \bar{\mathcal{Y}})| \leq M_{\mathcal{L}} \|\mathcal{Y} - \bar{\mathcal{Y}}\|. \tag{16}$$

Next, take the following mapping \mathcal{H} on \mathcal{X} :

$$\mathcal{H}\mathcal{Y}(t) = \mathcal{Y}_0(t) + \frac{1}{\Gamma(\vartheta)} \int_0^t (t-r)^{\vartheta-1} \mathcal{L}(r, \mathcal{Y}(r)) dr. \tag{17}$$

A solution to (12) exists under the conditions that C1 and C2 are fulfilled. To explore the solution of suggested model, we follow these steps:

Theorem 3. *The proposed campylobacteriosis system (8) exhibits at least one solution provided that the conditions C1 and C2 are fulfilled.*

Proof. First, apply the fixed-point theorem to prove the required findings. This theorem will be demonstrated through a delineation of four specific stages, as mentioned below:

P1: First, to establish the continuity of \mathcal{H} operator. The continuity of $\mathcal{L}(t, \mathcal{Y}(t))$ guaranteed from the continuity of \mathcal{Y}_i for $i = 1, 2, \dots, 5$. Next, considering $\mathcal{Y}_j, \mathcal{Y} \in \mathcal{X}$ in a way that $\mathcal{Y}_j \rightarrow \mathcal{Y}$, we have $\mathcal{H}\mathcal{Y}_j \rightarrow \mathcal{H}\mathcal{Y}$. Furthermore, let us take

$$\begin{aligned} \|\mathcal{H}\mathcal{Y}_j - \mathcal{H}\mathcal{Y}\| &= \max_{t \in [0, \tau]} \left| \frac{1}{\Gamma(\vartheta)} \int_0^t (t-r)^{\vartheta-1} \mathcal{Q}_j(r, \mathcal{Y}_j(r)) dr - \frac{1}{\Gamma(\vartheta)} \int_0^t (t-r)^{\vartheta-1} \mathcal{L}(r, \mathcal{Y}(r)) ds \right| \\ &\leq \max_{t \in [0, \tau]} \int_0^t \left| \frac{(t-r)^{\vartheta-1}}{\Gamma(\vartheta)} \right| |\mathcal{L}_j(r, \mathcal{Y}_j(r)) - \mathcal{L}(r, \mathcal{Y}(r))| dr \\ &\leq \frac{\tau^\vartheta M_{\mathcal{L}}}{\Gamma(\vartheta+1)} \|\mathcal{Y}_j - \mathcal{Y}\| \rightarrow 0 \text{ as } j \rightarrow \infty. \end{aligned} \tag{18}$$

The continuity of $\mathcal{H}\mathcal{Y}_j \rightarrow \mathcal{H}\mathcal{Y}$ is ensured from the continuity of \mathcal{L} , guaranteeing the continuity of \mathcal{H} .

P2: In this stage, we will prove the boundedness of \mathcal{H} . Take any $\mathcal{Y} \in \mathcal{X}$, and the subsequent conditions are met by means of the operator \mathcal{H} :

$$\begin{aligned} \|\mathcal{H}\mathcal{Y}\| &= \max_{t \in [0, \tau]} \left| \mathcal{Y}_0(t) + \frac{1}{\Gamma(\vartheta)} \int_0^t (t-r)^{\vartheta-1} \mathcal{L}(r, \mathcal{Y}(r)) dr \right| \\ &\leq |\mathcal{Y}_0| \max_{t \in [0, \tau]} \frac{1}{\Gamma(\vartheta)} \int_0^t |(t-r)^{\vartheta-1}| |\mathcal{L}(r, \mathcal{Y}(r))| dr \\ &\leq |\mathcal{Y}_0| + \frac{\tau^\vartheta}{\Gamma(\vartheta+1)} \{U_Z \|\mathcal{Y}\|^q + V_{\mathcal{L}}\}. \end{aligned} \tag{19}$$

We shall next prove that $\mathcal{H}(T)$ is bounded inside a bounded subset T of \mathcal{X} . Take $\mathcal{Y} \in T$, and by virtue of the bounded nature of S , one can find a non-negative value U satisfying

$$\|\mathcal{Y}\| \leq U, \forall \mathcal{Y} \in T. \tag{20}$$

In list of this, the outcome for any \mathcal{Y} within the set T is derived from the above expression as follows:

$$\|\mathcal{H}W\| \leq |\mathcal{Y}_0| + \frac{\tau^\vartheta}{\Gamma(\vartheta + 1)} [U_{\mathcal{L}} \|\mathcal{Y}\|^q + V_{\mathcal{L}}] \leq |\mathcal{Y}_0| + \frac{\tau^\vartheta}{\Gamma(\vartheta + 1)} [U_{\mathcal{L}}U^q + V_{\mathcal{L}}]. \tag{21}$$

Thus, the boundedness of $\mathcal{H}(T)$ is obtained.

P3: To prove equi-continuity, we assume t_1 and t_2 in $[0, \tau]$ in which $t_1 \geq t_2$. Subsequently, we obtain:

$$\begin{aligned} |\mathcal{H}\mathcal{Y}(t_1) - \mathcal{H}\mathcal{Y}(t_2)| &= \left| \frac{1}{\Gamma(\vartheta)} \int_0^{t_1} (t_1 - r)^{\vartheta-1} \|\mathcal{L}(r, \mathcal{Y}(r))\| dr \right. \\ &\quad \left. - \frac{1}{\Gamma(\vartheta)} \int_0^{t_2} (t_2 - r)^{\vartheta-1} \|\mathcal{L}(r, \mathcal{Y}(r))\| dr \right| \\ &\leq \left| \frac{1}{\Gamma(\vartheta)} \int_0^{t_1} (t_1 - r)^{\vartheta-1} - \frac{1}{\Gamma(\vartheta)} \int_0^{t_2} (t_2 - r)^{\vartheta-1} \right| \|\mathcal{L}(r, \mathcal{Y}(r))\| dr \\ &\leq \frac{\tau^\vartheta}{\Gamma(\vartheta + 1)} [U_{\mathcal{L}} \|\mathcal{Y}\|^q + V_{\mathcal{L}}] [t_1^\vartheta - t_2^\vartheta] \rightarrow 0 \text{ as } t_1 \rightarrow t_2. \end{aligned} \tag{22}$$

This ensures the relative compactness of $\mathcal{H}(T)$ using the Arzelà–Ascoli theorem:

P4: Finally, we examine the set outlined as follows:

$$\mathcal{E} = \{\mathcal{Y} \in \mathcal{X} : \mathcal{Y} = \lambda B\mathcal{Y}, \lambda \in (0, 1)\}. \tag{23}$$

For the boundedness of \mathcal{E} , let us suppose that \mathcal{Y} belongs to \mathcal{E} . For every t in $[0, \tau]$, the below condition fulfills:

$$\|\mathcal{Y}\| = \lambda \|B\mathcal{Y}\| \leq \lambda \left[|\mathcal{Y}_0| \frac{\tau^\vartheta}{\Gamma(\vartheta + 1)} [U_{\mathcal{L}} \|\mathcal{Y}\|^q + V_{\mathcal{L}}] \right]. \tag{24}$$

As a result, the boundedness of \mathcal{E} is ensured. Through the Schaefer’s theorem, there is a fixed point of the operator B . From this, there is at least one solution of the model (12) of campylobacteriosis.

Remark 1. If the condition C1 fulfills for $q = 1$, then it is possible to demonstrate Theorem 3 for $\frac{\tau^\vartheta U_{\mathcal{Z}}}{\Gamma(\vartheta+1)} < 1$.

Theorem 4. There is a unique solution of (12) of campylobacteriosis infection if the condition $\frac{\tau^\vartheta U_{\mathcal{Z}}}{\Gamma(\vartheta+1)} < 1$ is satisfied.

Proof. . To establish the proof, we utilize the well-known theorem of Banach’s contraction, take that both \mathcal{Y} and $\bar{\mathcal{Y}}$ belong to the set \mathcal{X} .

$$\begin{aligned} \|B\mathcal{Y} - B\bar{\mathcal{Y}}\| &\leq \max_{t \in [0, \tau]} \frac{1}{\Gamma(\vartheta)} \int_0^t (t - r)^{\vartheta-1} \|\mathcal{L}(r, \mathcal{Y}(r)) - \mathcal{L}(r, \bar{\mathcal{Y}}(r))\| dr \\ &\leq \frac{\tau^\vartheta U_{\mathcal{L}}}{\Gamma(\vartheta + 1)} \|\mathcal{Y} - \bar{\mathcal{Y}}\|. \end{aligned} \tag{25}$$

This implies that there is a unique fixed point for B , proving that the campylobacteriosis model (12) has a unique solution.

5. Stability analysis

Here, we aim to demonstrate Ulam-Hyers stability (UHS) for the proposed model of campylobacteriosis. The introduction of Ulam-Hyers stability dates back to Ulam in 1940 and was extended by Hyers [12]. The application of Ulam-Hyers stability to various academic domains has been explored by several researchers. The core theory can be summarized as follows:

Take $\mathcal{G} : \mathcal{X} \rightarrow \mathcal{X}$ such that

$$\mathcal{H}\mathcal{D} = \mathcal{D} \text{ for } \mathcal{D} \in \mathcal{X}. \quad (26)$$

Definition 4. The previously mentioned expression (26) qualifies as UHS if, for each solution \mathcal{D} within the set \mathcal{X} and for any given $\zeta > 0$, it is possible to identify.

$$\|\mathcal{D} - \mathcal{G}\mathcal{D}\| \leq \zeta, \quad (27)$$

for all t in the interval $[0, \tau]$. Furthermore, take a unique solution $\bar{\mathcal{D}}$ for the aforementioned upper bound (26), such that C_q is a positive value, and the following condition is satisfied.

$$\|\bar{\mathcal{D}} - \mathcal{D}\| \leq C_q \zeta, \quad (28)$$

for all t in the closed interval $[0, \tau]$.

Definition 5. If the solutions \mathcal{D} and $\bar{\mathcal{D}}$ of (26) satisfies the following

$$\|\bar{\mathcal{D}} - \mathcal{D}\| \leq \mathcal{L}(\zeta), \quad (29)$$

where zero have zero image, and $\mathcal{L} \in C(R, R)$. Then, the system (26) is generalized UHS.

Remark 2. If the solution denoted as $\bar{\mathcal{D}}$ belonging to the set \mathcal{X} holds (28). Then, the below satisfies for all t in $[0, \tau]$:

(a) $|\varpi(t)| \leq \zeta$, where $\varpi \in C([0, \tau]; R)$,

(b) $\mathcal{G}\bar{\mathcal{D}}(T) = \bar{\mathcal{D}} + \varpi(T)$.

Next, system (12) can be written in the following form after small changes:

$$\begin{cases} {}^C D_{0+}^{\vartheta} \mathcal{D}(t) = \mathcal{D}(t, \mathcal{D}(t)) + \varpi(t), \\ \mathcal{D}(0) = \mathcal{D}_0. \end{cases} \quad (30)$$

Lemma 2. Equation (30) satisfies the below

$$|\mathcal{D}(t) - T\mathcal{D}(t)| \leq a\zeta, \text{ where } a = \frac{\tau^{\vartheta}}{\Gamma(\vartheta + 1)}. \quad (31)$$

Applying Lemma (1) and considering Remark (2), one can prove it effortlessly.

Theorem 5. *If the inequality $\frac{\tau^\vartheta L\mathcal{D}}{\Gamma(\vartheta+1)} < 1$ holds, the solution to equation (12) demonstrates UHS and extending the concept of a generalizes UHS as per Lemma (2).*

Proof. . We consider the solutions \mathcal{D} and $\bar{\mathcal{D}} \in X$ of the system (12) as part of the necessary proof, thereby assuming:

$$\begin{aligned} |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| &= |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| \\ &\leq |\mathcal{D}(t) - T\bar{\mathcal{D}}(t)| \\ &\leq |\mathcal{D}(t) - T\bar{\mathcal{D}}(t)| \\ &\leq a\zeta + \frac{\tau^\xi L\mathcal{U}}{\Gamma(\xi+1)} |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| \\ &\leq \frac{a\zeta}{1 - \frac{\tau^\xi L\mathcal{U}}{\Gamma(\xi+1)}}. \end{aligned} \quad (32)$$

Due to this, the solution of (12) of campylobacteriosis is UHS and generalized UHS.

Definition 6. *If the below mentioned condition fulfills for any $\mathcal{D} \in \mathcal{X}$:*

$$\|\mathcal{D} - \mathcal{KV}\| \leq \Omega(t)\zeta, \text{ for } t \in [0, \tau], \quad (33)$$

Then, the solution of (26) exhibits Ulam-Hyers-Rassias stability (UHS). Here, Ω belongs to the space $C[[0, \tau], R]$ and ζ is a positive value. In the case where $C_q > 0$, a distinctive solution $\bar{\mathcal{D}}$ for the system (26) exists, meeting condition given as follows:

$$\|\bar{\mathcal{D}} - \mathcal{D}\| \leq C_q \Omega(t)\zeta, \quad \forall t \in [0, \tau]. \quad (34)$$

Definition 7. *Consider the unique solution $\bar{\mathcal{D}}$, and let \mathcal{D} denote any alternative solution to the equation (26), where*

$$\|\bar{\mathcal{D}} - \mathcal{D}\| \leq C_{q,\Omega} \Omega(t)\zeta, \quad (35)$$

whereas t resides in the interval $[0, \tau]$ and $\Omega \in D[[0, \tau], R]$ such that $C_{q,\Omega}$ and $\zeta > 0$. Consequently, it indicates that the solution to (26) is a generalized UHS.

Remark 3. *Take $\bar{\mathcal{D}} \in X$, this solution satisfies (28) for all t in the interval $[0, \tau]$ if*

(a) $|\varpi(t)| \leq \zeta\Omega(t)$, where $\varpi(t) \in \mathcal{C}([0, \tau]; R)$

(b) $\mathcal{K}\bar{\mathcal{D}}(t) = \bar{\mathcal{D}} + \varpi(t)$.

Lemma 3. *The system described in (2) with perturbation satisfies the subsequent conditions:*

$$|\mathcal{D}(t) - T\mathcal{D}(T)| \leq a\Omega(t)\zeta, \text{ in which } a = \frac{\tau^\vartheta}{\Gamma(\vartheta+1)}. \quad (36)$$

This can be easily proved with the help of Remark (3) and Lemma (1).

Theorem 6. *The solution to (12) corresponds to the UHS and generalized UHS based on Lemma (3), provided that $\frac{\tau^\vartheta L_{\mathcal{U}}}{\Gamma(\vartheta+1)} < 1$ is fulfilled.*

Proof. Assume one can find a unique solution $\bar{\mathcal{D}} \in \mathcal{X}$, and for any alternative solution $\mathcal{D} \in \mathcal{X}$ of (12), the below satisfies:

$$\begin{aligned} |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| &= |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| \\ &\leq |\mathcal{D}(t) - T\bar{\mathcal{D}}(t)| \\ &\leq |\mathcal{D}(t) - T\bar{\mathcal{D}}(t)| \\ &\leq a\Omega(t)\zeta + \frac{\tau^\vartheta L_{\mathcal{D}}}{\Gamma(\vartheta+1)} |\mathcal{D}(t) - \bar{\mathcal{D}}(t)| \\ &\leq \frac{a\Omega(t)\zeta}{1 - \frac{\tau^\vartheta L_{\mathcal{D}}}{\Gamma(\vartheta+1)}}. \end{aligned} \quad (37)$$

Due to this, the solution to the equation (12) corresponds to UHS and generalized UHS.

6. Numerical scheme for the system

Numerical and analytical methods are critical tools for solution analysis in mathematical modeling. They provide powerful approaches for approximating solutions to complex problems, thereby enhancing both accuracy and reliability while offering flexibility in their application. Here, a numerical method is introduced to solve the proposed fractional model (8) of campylobacteriosis. The subsequent process is presented,

$${}^C D_t^\vartheta \mathcal{F}(t) = \bar{h}(t, \mathcal{F}(t)). \quad (38)$$

Utilizing the fundamental theorem on the equation represented by (38), we acquire:

$$\mathcal{F}(t) - \mathcal{F}(0) = \frac{1}{\Gamma(\vartheta)} \int_0^t \bar{h}(\eta, \mathcal{F}(\eta))(t - \eta)^{\vartheta-1} d\eta, \quad (39)$$

therefore, at time $t = t_{n+1}$, $n = 0, 1, \dots$, the below is acquired:

$$\mathcal{F}(t_{n+1}) - \mathcal{F}(0) = \frac{1}{\Gamma(\vartheta)} \int_0^{t_{n+1}} (t_{n+1} - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt, \quad (40)$$

and

$$\bar{h}(t_n) - \bar{h}(0) = \frac{1}{\Gamma(\vartheta)} \int_0^{t_n} (t_n - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt. \quad (41)$$

From (41) and (40), we have

$$\mathcal{F}(t_{n+1}) = \mathcal{F}(t_n) + \underbrace{\frac{1}{\Gamma(\vartheta)} \int_0^{t_{n+1}} (t_{n+1} - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt}_{\mathcal{A}_{\vartheta,1}}$$

$$-\underbrace{\frac{1}{\Gamma(\vartheta)} \int_0^{t_n} (t_n - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt}_{\mathcal{A}_{\vartheta,2}}, \quad (42)$$

where

$$\mathcal{A}_{\vartheta,1} = \frac{1}{\Gamma(\vartheta)} \int_0^{t_{n+1}} (t_{n+1} - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt, \quad (43)$$

and

$$\mathcal{A}_{\vartheta,2} = \frac{1}{\Gamma(\vartheta)} \int_0^{t_n} (t_n - t)^{\vartheta-1} \bar{h}(t, \mathcal{F}(t)) dt. \quad (44)$$

By employing the Lagrange approximation with respect to $\bar{h}(t, \mathcal{F}(t))$, we derive:

$$\begin{aligned} \mathbf{P}(t) &\simeq \frac{t - t_{n-1}}{t_n - t_{n-1}} \bar{h}(t_n, \mathcal{F}_n) + \frac{t - t_n}{t_{n-1} - t_n} \bar{h}(t_{n-1}, \mathcal{F}_{n-1}) \\ &= \frac{f(t_n, \mathcal{F}_n)}{h} (t - t_{n-1}) - \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h} (t - t_n). \end{aligned} \quad (45)$$

Utilizing the expression mentioned above yields

$$\begin{aligned} \mathcal{A}_{\vartheta,1} &= \frac{\bar{h}(t_n, \mathcal{F}_n)}{h\Gamma(\vartheta)} \int_0^{t_{n+1}} (t_{n+1} - t)^{\vartheta-1} (t - t_{n-1}) dt \\ &\quad - \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h\Gamma(\vartheta)} \int_0^{t_{n+1}} (t_{n+1} - t)^{\vartheta-1} (t - t_n) dt. \end{aligned} \quad (46)$$

After further simplification, we have

$$\begin{aligned} \mathcal{A}_{\vartheta,1} &= \frac{\bar{h}(t_n, \mathcal{F}_n)}{h\Gamma(\vartheta)} \left[\frac{2h}{\vartheta} t_{n+1}^{\vartheta} - \frac{t_{n+1}^{\vartheta+1}}{\vartheta+1} \right] \\ &\quad - \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h\Gamma(\vartheta)} \left[\frac{h}{\vartheta} t_{n+1}^{\vartheta} - \frac{1}{\vartheta+1} t_{n+1}^{\vartheta+1} \right]. \end{aligned} \quad (47)$$

Similarly, the below is obtained

$$\begin{aligned} \mathcal{A}_{\vartheta,2} &= \frac{1}{\Gamma(\vartheta)} \int_0^{t_n} (t_n - t)^{\vartheta-1} \left[\frac{\bar{h}(t_n, \mathcal{F}_n)}{h} (t - t_{n-1}) \right. \\ &\quad \left. - \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h} (t - t_n) \right] dt. \end{aligned} \quad (48)$$

Continuing with the simplification process, we arrive at the following:

$$\mathcal{A}_{\vartheta,2} = \frac{\bar{h}(t_n, \mathcal{F}_n)}{h\Gamma(\vartheta)} \left[\frac{h}{\vartheta} t_n^{\vartheta} - \frac{t_n^{\vartheta+1}}{\vartheta+1} \right]$$

$$+ \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h\Gamma(\vartheta)} \left[\frac{1}{\vartheta + 1} t_n^{\vartheta+1} \right]. \tag{49}$$

Substituting (48) and (49) into (42), we have

$$\begin{aligned} \mathcal{F}(t_{n+1}) = & \mathcal{F}(t_n) + \frac{\bar{h}(t_n, \mathcal{F}_n)}{h\Gamma(\vartheta)} \left[\frac{2ht_{n+1}^\vartheta}{\vartheta} - \frac{t_{n+1}^{\vartheta+1}}{\vartheta + 1} + \frac{h}{\vartheta} t_n^\vartheta - \frac{t_{n+1}^{\vartheta+1}}{\vartheta + 1} \right] \\ & + \frac{\bar{h}(t_{n-1}, \mathcal{F}_{n-1})}{h\Gamma(\vartheta)} \left[-\frac{h}{\vartheta} t_{n+1}^\vartheta + \frac{t_{n+1}^{\vartheta+1}}{\vartheta + 1} + \frac{t_n^{\vartheta+1}}{\vartheta + 1} \right]. \end{aligned} \tag{50}$$

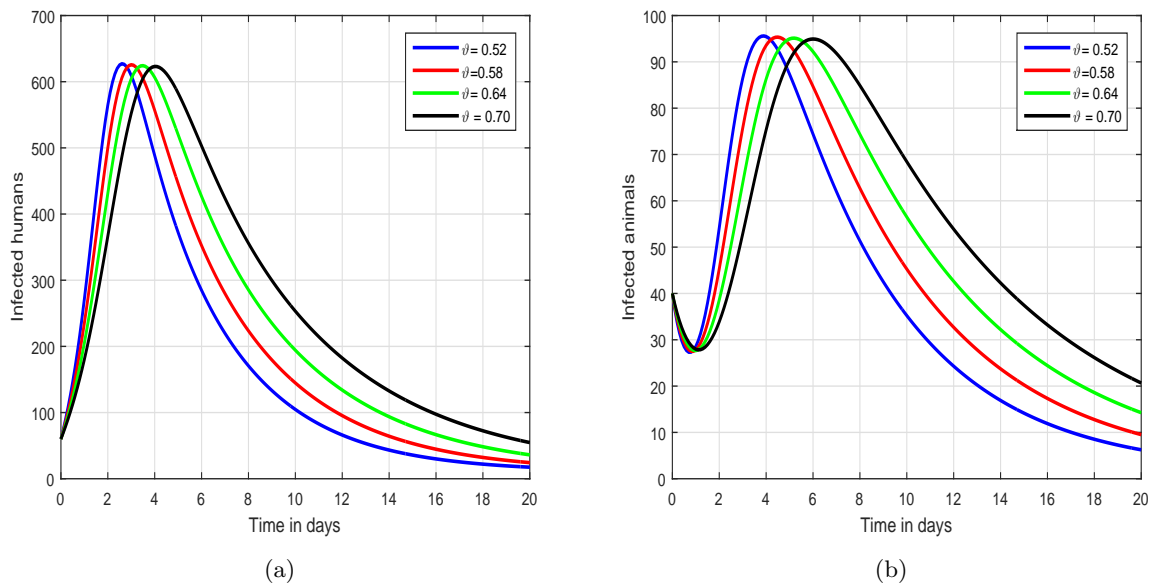


Figure 2: Visualization of the tracking paths of the compartments of the campylobacteriosis model (8) with different values of ϑ , i.e., $\vartheta = 0.52, 0.58, 0.64, 0.70$.

We examined the dynamical behavior of our campylobacteriosis infection model (8) using the numerical scheme outlined above. Our primary objective is to utilize these findings to show how the factors of the system influence the dynamics of campylobacteriosis. Through our analysis, we aim to propose effective control strategies that can lower the prevalence of campylobacteriosis. Time series analysis of an epidemic model involves examining and interpreting data collected over time to understand the patterns and dynamics of the epidemic. This typically includes tracking the spread of the disease, identifying factors that influence its transmission, and making predictions for future trends. In our simulations, We have demonstrated the variation in the infected strengths of humans and animals. The numerical simulations involve assumed values for the state variables and parameters.

In the initial simulation presented in Figure 2 and Figure 3, we demonstrated the influence of the fractional order on the population of infected individuals in the proposed

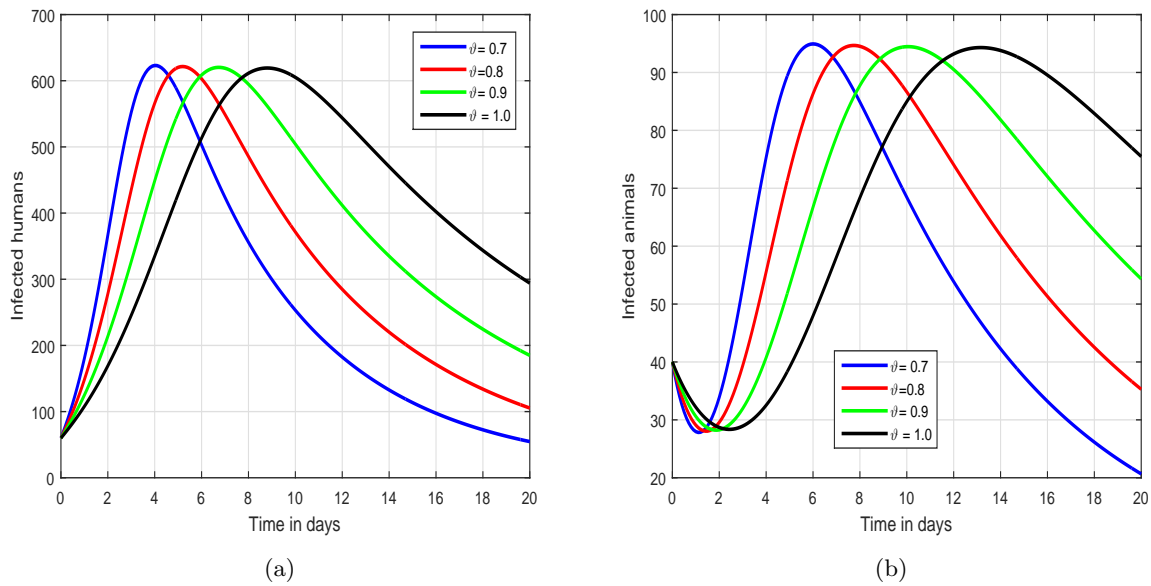


Figure 3: Graphical view analysis of the dynamics of the campylobacteriosis model (8) with the variation of ϑ , i.e., $\vartheta = 0.70, 0.80, 0.90, 1.00$.

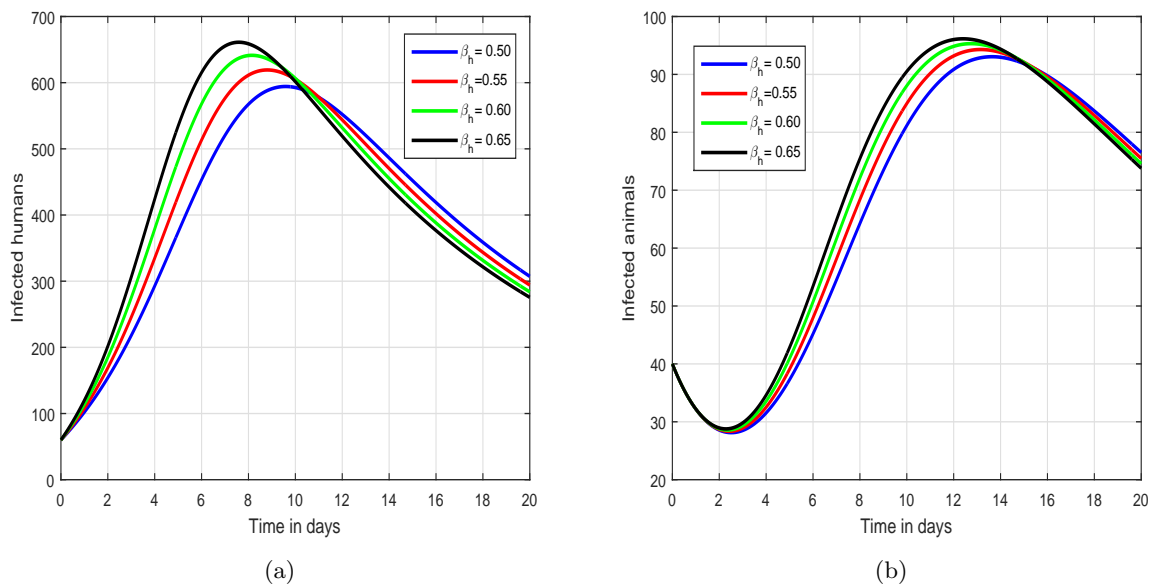


Figure 4: Visualization of the progression route of the campylobacteriosis model (8) with various values transmission rate β_h , i.e., $\beta_h = 0.50, 0.55, 0.60, 0.65$.

model of campylobacteriosis. We assumed different values of the parameter ϑ in these figures with comparison analysis to the ordinary system. In Figure 2, the values of ϑ is considered to be $\vartheta = 0.52, 0.58, 0.64$ and 0.70 while in Figure 3, the values of ϑ is assumed

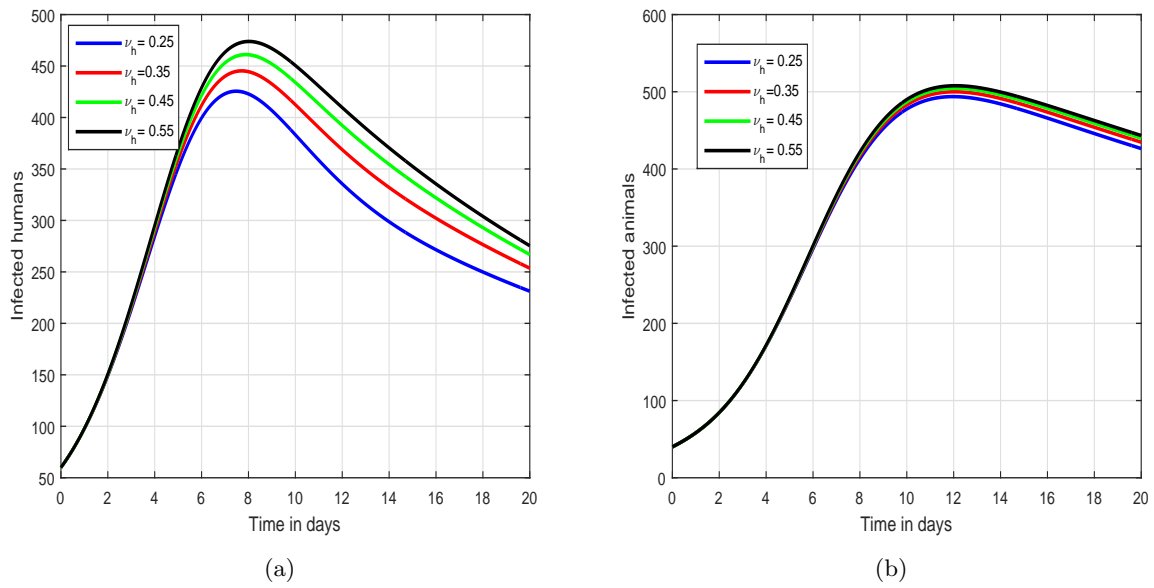


Figure 5: Numerical investigation of the model (8) of campylobacteriosis with various values losing rate of immunity ν_h , i.e., $\nu_h = 0.25, 0.35, 0.45, 0.55$.

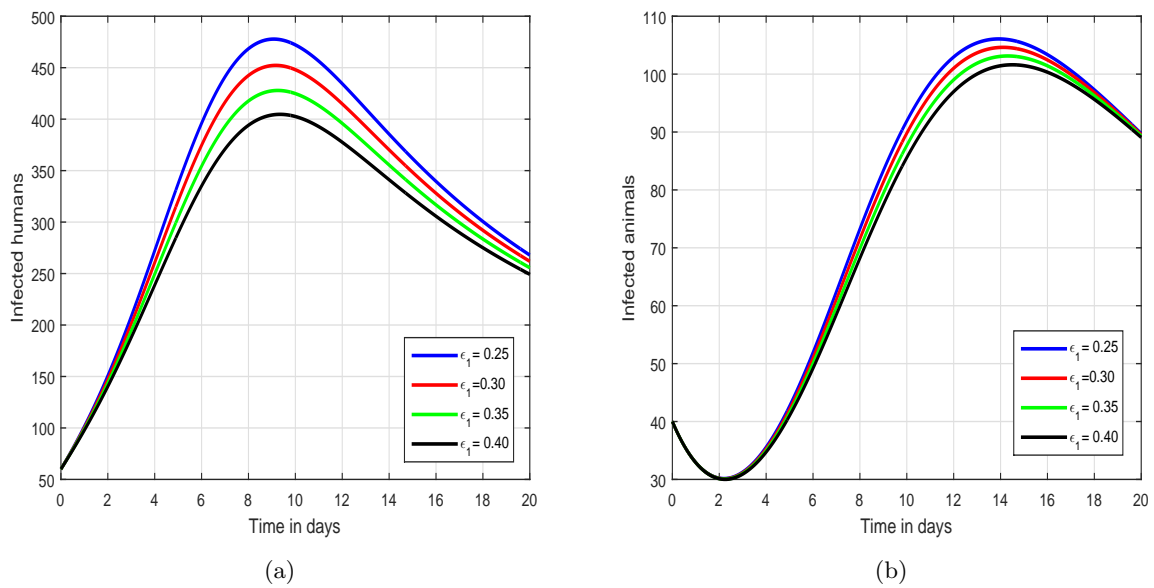


Figure 6: Graphical view analysis of campylobacteriosis model (8) with the efficacy of drugs in treatment ϵ_1 , i.e., $\epsilon_1 = 0.25, 0.30, 0.35, 0.40$.

to be $\vartheta = 0.70, 0.80, 0.90$ and 1.0 . The effect of classical derivative and fractional derivative can be seen in 3 which shows that fractional systems are more flexible and different values can be considered rather than one. We observed that the index of memory has a significant

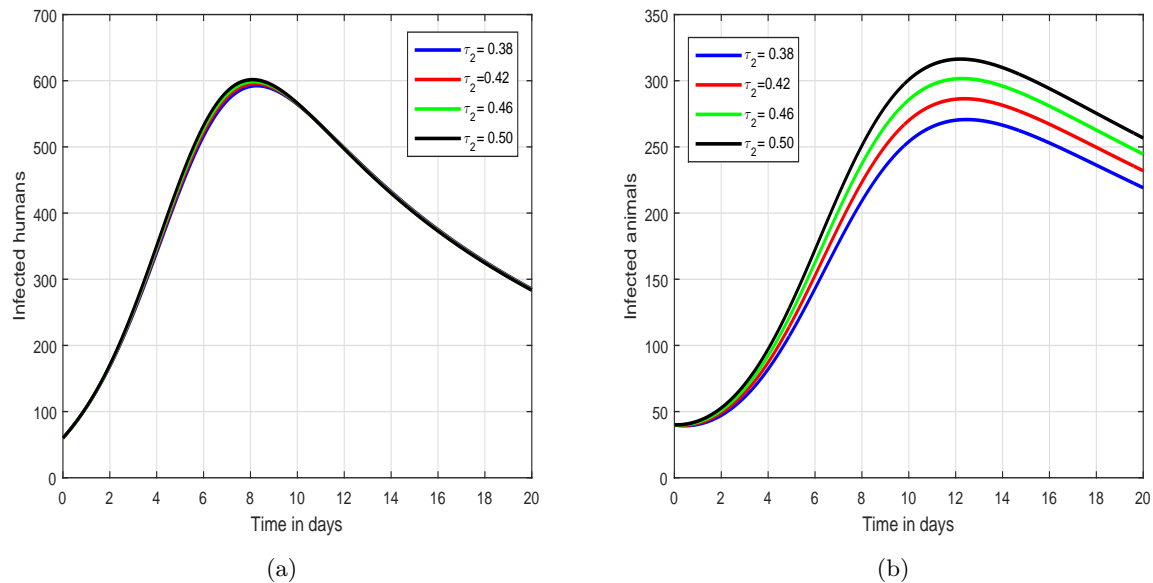


Figure 7: Visualization of the tracking path of the model (8) of campylobacteriosis with different values of τ_2 , i.e., $\tau_2 = 0.38, 0.42, 0.46, 0.50$.

and attractive effect on the dynamics of infected individuals. Furthermore, the decrease in this system parameter can lead to a decline in the infection levels within the society. It is evident that the number of infected individuals can be influenced by controlling the memory index. Consequently, policymakers are encouraged to consider the memory index as a viable tool for controlling and preventing the infection. In Figure 4, we have shown the impact of transmission rate β_h on the dynamics of infected individuals of the system. We assumed the values of β_h to be 0.50, 0.55, 0.60 and 0.65 in the second simulation. Observably, this factor dangerous and elevates the risk of infection within society. Hence, we warn health officials against this critical factor which increase the risk of infection.

In Figure 5, showed the consequences of lose rate of immunity on the transmission dynamics of campylobacteriosis. We assumed the value of ν_h to be 0.25, 0.35, 0.45 and 0.55. In this simulation, we can see that this input factor is dangerous for the infected humans and increase the level of infection. On the basis of this result, we can say that ν_a is also dangerous which make the control more difficult in animals. In Figure 6, we have shown the role of the efficacy of drug during treatment on the dynamics of the infection. This factor exerts a positive impact on the system, contributing to a reduction in the infection level. Figure 7 demonstrates the influence of the input parameter τ_2 on the dynamics of campylobacteriosis. The values of τ_2 are assumed to be 0.38, 0.42, 0.46 and 0.50 in this simulation. This factor exerts a beneficial effect on the system, leading to a decrease in the infection levels.

7. Concluding remarks

In this research, we formulated the dynamics of campylobacteriosis with drug resistance in both humans and animals using a fractional framework. We presented the fundamental concepts of fractional derivatives to analyze the proposed model of infection. Both qualitative and quantitative analyses of the dynamics were conducted. The fixed-point theorem was employed to investigate the existence and uniqueness of solutions, while the stability of the system was established through analytical techniques. Additionally, we introduced a numerical scheme to visualize the solution pathways of the infection and to evaluate the influence of various factors within the system. Our findings illustrated the dynamics of campylobacteriosis with drug resistance, emphasizing the effects of different factors on infection levels. In this work, we identified key factors that are essential for effective disease control and management. In future research, we plan to examine the impact of impulsive vaccination on the transmission dynamics of campylobacteriosis to mitigate the economic burden of the infection.

Acknowledgements

Acknowledgments The researchers would like to thank the Deanship of Graduate Studies and Scientific Research at Qassim University for financial support (QU-APC-2024-9/1).

References

- [1] Hamadjam Abboubakar, Rubin Fandio, Brandon Satsa Sofack, and Henri Paul Ekobena Fouda. Fractional dynamics of a measles epidemic model. *Axioms*, 11(8):363, 2022.
- [2] Fayyaz Ahmad, Kifayat Ullah, Junaid Ahmad, Ahmad Aloqaily, and Nabil Mlaiki. Computational analysis of a novel iterative scheme with an application. *Computation*, 12(9):192, 2024.
- [3] Ghada Ahmed. Analysis and applications of the chaotic hyperbolic memristor model with fractional order derivative. *European Journal of Pure and Applied Mathematics*, 17(2):835–851, 2024.
- [4] Tosin Akin Akinmolayan, Jude Oluwapelumi Alao, Eunice Damilola Wilkie, Daniel Abayomi Odeyemi, Taofikat Olatundun Akintoyese, and Abosede Yetunde Owolabi. Strategies for controlling campylobacter in poultry production: A comprehensive review of challenges and potential solutions. *South Asian Journal of Research in Microbiology*, 17(2):50–61, 2023.
- [5] SQ Amin, HJ Mahmood, and HK Zorab. Campylobacteriosis. *One Health Triad*, 2:87–93, 2023.

- [6] Hannah K Bolinger. *Emerging Trends of Antibiotic Resistance and Risk Factors for Campylobacter spp. in Commercially Produced Turkey Flocks*. North Carolina State University, 2017.
- [7] A Ait Brahim, A El Hajaji, K Hilal, and J El Ghordaf. On a novel fractional calculus and its applications to well-known problems. *European Journal of Pure and Applied Mathematics*, 17(2):1155–1167, 2024.
- [8] Furaha Michael Chuma and Edward Kanuti Ngailo. Mathematical analysis of campylobacteriosis disease model in human with saturated incidence rate and treatment. *Mathematics Open*, 3:2350011, 2024.
- [9] N Deborah Friedman, Elizabeth Temkin, and Yehuda Carmeli. The negative impact of antibiotic resistance. *Clinical microbiology and infection*, 22(5):416–422, 2016.
- [10] Andrzej Granas, James Dugundji, Andrzej Granas, and James Dugundji. Elementary fixed point theorems. *Fixed point theory*, pages 9–84, 2003.
- [11] Tharmalingam Gunasekar, Shanmugam Manikandan, Vedyappan Govindan, Junaid Ahmad, Walid Emam, and Isra Al-Shbeil. Symmetry analyses of epidemiological model for monkeypox virus with atangana–baleanu fractional derivative. *Symmetry*, 15(8):1605, 2023.
- [12] Donald H Hyers. On the stability of the linear functional equation. *Proceedings of the National Academy of Sciences*, 27(4):222–224, 1941.
- [13] Asif Jan, Rashid Jan, Hassan Khan, MS Zobaer, and Rasool Shah. Fractional-order dynamics of rift valley fever in ruminant host with vaccination. *Commun. Math. Biol. Neurosci.*, 2020:Article–ID, 2020.
- [14] Rashid Jan, Evren Hınçal, Kamyar Hosseini, Normy Norfiza Abdul Razak, Thabet Abdeljawad, and MS Osman. Fractional view analysis of the impact of vaccination on the dynamics of a viral infection. *Alexandria Engineering Journal*, 102:36–48, 2024.
- [15] Rashid Jan and Asif Jan. Msgdtm for solution of fractional order dengue disease model. *International Journal of Science and Research*, 6(3):1140–1144, 2017.
- [16] Rashid Jan, Zahir Shah, Wejdan Deebani, and Ebraheem Alzahrani. Analysis and dynamical behavior of a novel dengue model via fractional calculus. *International Journal of Biomathematics*, 15(06):2250036, 2022.
- [17] Kilbas, Anatoliĭ Aleksandrovich and Srivastava, Hari M and Trujillo, Juan J. *Theory and applications of fractional differential equations*, volume 204. Elsevier, 2006.
- [18] Mikayla Plishka, Jan M Sargeant, Charlotte Winder, and Amy L Greer. Modelling the introduction and transmission of campylobacter in a north american chicken flock. *Zoonoses and Public Health*, 69(1):23–32, 2022.

- [19] Randall S Singer, Louis A Cox Jr, James S Dickson, H Scott Hurd, Ian Phillips, and Gay Y Miller. Modeling the relationship between food animal health and human foodborne illness. *Preventive veterinary medicine*, 79(2-4):186–203, 2007.
- [20] Bernadeta Szczepańska, Piotr Kamiński, Małgorzata Andrzejewska, Dorota Śpica, Edmund Kartanas, Werner Ulrich, Leszek Jerzak, Mariusz Kasprzak, Marcin Bocheński, and Jacek J Klawe. Prevalence, virulence, and antimicrobial resistance of campylobacter jejuni and campylobacter coli in white stork ciconia ciconia in poland. *Foodborne pathogens and disease*, 12(1):24–31, 2015.
- [21] Tao-Qian Tang, Rashid Jan, Hassan Ahmad, Zahir Shah, Narcisa Vrinceanu, and Mihaela Racheriu. A fractional perspective on the dynamics of hiv, considering the interaction of viruses and immune system with the effect of antiretroviral therapy. *Journal of Nonlinear Mathematical Physics*, 30(4):1327–1344, 2023.
- [22] Tao-Qian Tang, Zahir Shah, Rashid Jan, Wejdan Deebani, and Meshal Shutaywi. A robust study to conceptualize the interactions of cd4+ t-cells and human immunodeficiency virus via fractional-calculus. *Physica Scripta*, 96(12):125231, 2021.
- [23] Chris A Whitehouse, Shaohua Zhao, and Heather Tate. Antimicrobial resistance in campylobacter species: mechanisms and genomic epidemiology. In *Advances in applied microbiology*, volume 103, pages 1–47. Elsevier, 2018.
- [24] Ongwae H Zachariah, Mwamburi A Lizzy, Kakai Rose, and Mutuku M Angela. Multiple drug resistance of campylobacter jejuni and shigella isolated from diarrhoeic children at kapsabet county referral hospital, kenya. *BMC Infectious Diseases*, 21:1–8, 2021.