Hindawi Publishing Corporation
Abstract and Applied Analysis
Volume 2014, Article ID 136263, 9 pages
http://dx.doi.org/10.1155/2014/136263

Research Article

On Fractional SIRC Model with *Salmonella* Bacterial Infection

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Received 3 March 2014; Accepted 25 March 2014; Published 22 April 2014

Academic Editor: Ali H. Bhrawy

We propose a fractional order SIRC epidemic model to describe the dynamics of *Salmonella* bacterial infection in animal herds. The infection-free and endemic steady sates, of such model, are asymptotically stable under some conditions. The basic reproduction number $\mathcal{R}_0$ is calculated, using next-generation matrix method, in terms of contact rate, recovery rate, and other parameters in the model. The numerical simulations of the fractional order SIRC model are performed by Caputo's derivative and using unconditionally stable implicit scheme. The obtained results give insight to the modelers and infectious disease specialists.

1. Introduction

During the past three decades, the subject of fractional calculus has gained popularity and importance, mainly due to its demonstrated applications in numerous diverse and widespread fields of science and engineering. For example, fractional calculus has been successfully applied to system biology, physics, chemistry and biochemistry, hydrology, medicine, and finance (see, e.g., [1–10] and the references therein). In many cases, the fractional order differential/integral equations models are more consistent with the real phenomena than the integer-order models because the fractional derivatives and integrals enable the description of the memory and hereditary properties inherent in various materials and processes. Hence, there is a growing need to study and use the fractional order differential and integral equations. However, analytical and closed solutions of these types of fractional equations cannot generally be obtained. As a consequence, approximate and numerical techniques are playing important role in identifying the solution behavior of such fractional equations and exploring their applications (see, e.g., [9, 11, 12] and the references therein).

We recall that the *Salmonella* infection is a major zoonotic disease which is transmitted between humans and other animals. Most persons infected with *Salmonella* develop diarrhea, fever, and abdominal cramps 12 to 72 hours after infection. The illness usually lasts 4 to 7 days, and most persons recover without treatment. However, in some persons, the diarrhea may be so severe that the patient needs to be hospitalized. *Salmonella* live in the intestinal tracts of humans and other animals, including birds. *Salmonella* are usually transmitted to humans by eating foods contaminated with animal feces. Contaminated foods usually look and smell normal. Contaminated foods are often of animal origin, such as beef, poultry, milk, or eggs, but any food, including vegetables, may become contaminated [13]. Therefore, *Salmonella* is considered as a serious problem for the public health throughout the world. There are no doubts that mathematical modeling of *Salmonella* bacterial infection plays an important role in gaining understanding of the transmission of the disease in specific environment and in predicting the behavior of any outbreak. Furthermore, mathematical analysis leads to determining the nature of equilibrium states and to suggesting recommended actions

to be taken by decision makers to control the spreading of the disease. The objective of this work is to adopt the fractional order epidemic model to describe the dynamics of *Salmonella* infections in animal herds.

A large amount of work done on modelling biological systems has been restricted to integer-order ordinary (or delay) differential equations (see, e.g., [14–19]). In [20], the authors proposed the classical *Susceptible-Infected-Recovered* (SIR) model. The authors in [21] introduced a new compartment into SIR model, which is called cross-immune compartment to be called SIRC model. The new compartment cross-immune $C(t)$ describes an intermediate state between the fully susceptible $S(t)$ and the fully protected $R(t)$ one. Recently, the fractional order SIRC model of influenza, a disease in human population, was discussed in [22]. In the present paper, we consider the fractional order SIRC model associated with evolution of *Salmonella* bacterial infection in animal herds. However, we will take into account the disease-induced mortality rate m in the model. Qualitative behavior of the fractional order SRIC model is then investigated. Numerical simulations of the fractional order SRIC model are provided to demonstrate the effectiveness of the proposed method by using implicit Euler's method.

We first give the definition of fractional order integration and fractional order differentiation [23–25]. Let $L^1 = L^1[a,b]$ be the class of Lebesgue integrable functions on $[a,b]$, $a < b < \infty$.

Definition 1. The fractional integral of order $\nu \in \mathbb{R}^+$ of the function $f(t), t > 0$ ($f : \mathbb{R}^+ \to \mathbb{R}$) is defined by

$$I_a^\nu f(t) = \frac{1}{\Gamma(\nu)} \int_a^t (t-s)^{\nu-1} f(s)\, ds, \quad t > 0. \tag{1}$$

However, the fractional derivative of order $\alpha \in (n-1, n)$ of $f(t)$ is defined by two ways.

(i) Riemann-Liouville fractional derivative: take fractional integral of order $(n-\alpha)$ and then take nth derivative:

$$D_a^\alpha f(t) = D_a^n I_a^{n-\alpha} f(t), \quad D_*^n = \frac{d^n}{dt^n}, \quad n = 1, 2, \ldots. \tag{2}$$

(ii) Caputo's fractional derivative: take nth derivative and then take a fractional integral of order $(n-\alpha)$:

$$D_a^\alpha f(t) = I_a^{n-\alpha} D_a^n f(t), \quad n = 1, 2, \ldots. \tag{3}$$

We notice that the definition of time-fractional derivative of a function $f(t)$ at $t = t_n$ involves an integration and calculating time-fractional derivative that requires all the past history, that is, all the values of $f(t)$ from $t = 0$ to $t = t_n$. Caputo's definition, which is a modification of the Riemann-Liouville definition, has the advantage of dealing properly with initial value problems. For more properties of the fractional derivatives and integrals, we refer to [8, 9, 24, 25] and references therein. The generalized mean value theorem is defined in the following Remark [26].

Remark 2. (i) Suppose that $f(t) \in C[a,b]$ and $D_*^\alpha f(t) \in C(a,b]$ for $0 < \alpha \le 1$; then we have

$$f(t) = f(a) + \frac{1}{\Gamma(\alpha)} D_*^\alpha f(\xi)\,(t-a)^\alpha, \quad \text{with } a < \xi < t \ \forall t \in (a,b]. \tag{4}$$

(ii) If (i) holds and $D_*^\alpha f(t) \ge 0 \ \forall t \in [a,b]$, then $f(t)$ is nondecreasing for each $t \in [a,b]$. If $D_*^\alpha f(t) \le 0 \ \forall t \in [a,b]$, then $f(t)$ is nonincreasing for each $t \in [a,b]$.

2. The SIRC Epidemic Model

Assume that the *Salmonella* infection spreads in animal herds which are grouped as four compartments, according to their infection status: $S(t)$ is the proportion of susceptible individuals at time t (individuals that do not have the bacterial infection), $I(t)$ is the proportion of infected individuals (that have the bacterial infection), $R(t)$ is the proportion of recovered individuals (that recovered from the infection and have temporary immunity), and $C(t)$ is the proportion of cross-immune individuals at time t. The total number of animals in the herd is given by $N = S + I + R + C$. We consider that initially all the animals are susceptible to the infection. Once infected, a susceptible individual leaves the susceptible compartment and enters the infectious compartment where it then becomes infectious. The infected animals pass into the recovered compartment. The individuals who have recovered from the disease have temporary immunity and grouped into $C(t)$ compartment. Therefore, we consider that the disease transmission model consists of nonnegative initial conditions together with system of equations:

$$\begin{aligned}
\dot{S}(t) &= \mu N + \eta C(t) - (\beta I(t) + \mu) S(t), \\
\dot{I}(t) &= \beta S(t) I(t) + \sigma\beta C(t) I(t) - (\theta + m + \mu) I(t), \\
\dot{R}(t) &= (1-\sigma)\beta C(t) I(t) + \theta I(t) - (\mu + \delta) R(t), \\
\dot{C}(t) &= \delta R(t) - \beta C(t) I(t) - (\eta + \mu) C(t).
\end{aligned} \tag{5}$$

Here, parameter μ denotes the mortality rate in every compartment and is assumed to be equal to the rate of newborns in the population. β is the contact rate and also called transmission from susceptible to infected. η^{-1} is the cross-immune period, θ^{-1} is the infectious period, δ^{-1} is the total immune period, and σ is the fraction of the exposed cross-immune individuals who are recruited in a unit time into the infective subpopulation [21, 27]. We also assume that the disease induces mortality rate m; see the diagram of Figure 1.

2.1. Fractional Order of SIRC Epidemic Model. Although a large number of work has been done in modeling the dynamics of epidemiological diseases, it has been restricted to integer-order (delay) differential equations. In recent years, it has turned out that many phenomena in different fields can be described very successfully by models using *fractional order differential equations* (FODEs) [1, 5, 28]. Now, we introduce

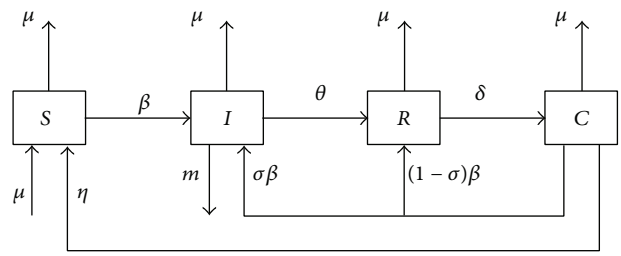

Figure 1: Schematic diagram of SIRC model.

fractional order into model (5) and assume that $s(t) = S(t)/N$, $i(t) = S(t)/N$, $r(t) = R(t)/N$, $c(t) = C(t)/N$, where N is the total number of population. Then the model takes the form

$$\begin{aligned} D^{\alpha_1} s(t) &= \mu + \eta c(t) - (\beta i(t) + \mu) s(t), \\ D^{\alpha_2} i(t) &= \beta s(t) i(t) + \sigma\beta c(t) i(t) - (\theta + m + \mu) i(t), \\ D^{\alpha_3} r(t) &= (1-\sigma)\beta c(t) i(t) + \theta i(t) - (\mu + \delta) r(t), \\ D^{\alpha_4} c(t) &= \delta r(t) - \beta c(t) i(t) - (\eta + \mu) c(t) \end{aligned} \tag{6}$$

with initial conditions $s(0) = s_0$, $i(0) = i_0$, and $r(0) = r_0$.

2.2. Stability Criteria for the Fractional Order SIRC Model. In model (6), assume that $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_4 = \alpha$. Then, to find the equilibria, we put $D^\alpha s = D^\alpha i = D^\alpha r = D^\alpha c = 0$.

Consider

$$\mathscr{E}_0 = (1, 0, 0, 0), \qquad \mathscr{E}_+ = (s^*, i^*, r^*, c^*), \tag{7}$$

where

$$\begin{aligned} s^* &= \frac{\theta + m + \mu}{\beta} - \sigma\left(\frac{\delta\theta i^*}{(\mu + \delta\sigma)\beta i^* + (\mu + \delta)(\mu + \eta)}\right), \\ r^* &= \frac{\theta i^* (\beta i^* + \eta + \mu)}{(\mu + \delta\sigma)\beta i^* + (\mu + \delta)(\mu + \eta)}, \\ c^* &= \frac{\theta\delta i^*}{(\mu + \delta\sigma)\beta i^* + (\mu + \delta)(\mu + \eta)}. \end{aligned} \tag{8}$$

The positive endemic equilibrium $\mathscr{E}_+ = (s^*, i^*, r^*, c^*)$ satisfies (6) and i^* is the positive root of $A_1 i^{*2} + A_2 i^* + A_3$, where

$$\begin{aligned} A_1 &= -\beta^2 [m(\mu + \delta\sigma) + \mu(\theta + \mu + \delta\sigma)], \\ A_2 &= \beta [\beta\mu(\mu + \delta\sigma) + \eta\theta\delta - (\theta + m + \mu) \\ &\quad \times [(\mu + \delta)(\mu + \eta) + \mu(\mu + \delta\sigma)] + \mu\sigma\delta\theta], \\ A_3 &= \beta\mu(\mu + \delta)(\mu + \eta)\left[1 - \left(\frac{\theta + m + \mu}{\beta}\right)\right]. \end{aligned} \tag{9}$$

The Jacobian matrix of model (6) is

$$J = \begin{pmatrix} -\beta i(t) - \mu & -\beta s(t) & 0 & \eta \\ \beta i(t) & \beta s(t) + \sigma\beta c(t) - (\theta + m + \mu) & 0 & \sigma\beta i(t) \\ 0 & (1-\sigma)\beta c(t) + \theta & -(\mu + \delta) & (1-\sigma)\beta i(t) \\ 0 & -\beta c(t) & \delta & -\beta i(t) - (\eta + \mu) \end{pmatrix}. \tag{10}$$

2.3. The Reproduction Number $\mathscr{R}_0$. The basic reproduction number (the number of individuals infected by a single infected individual placed in a totally susceptible population) $\mathscr{R}_0$ that includes the indirect transmission may be obtained using next-generation matrix method. The spectral radius of the next-generation matrix (FV^{-1}), which is the dominant eigenvalue of the same matrix, gives the value of $\mathscr{R}_0$ [29]. Then, the basic reproductive number $\mathscr{R}_0$ is obtained by the form

$$\mathscr{R}_0 = \rho\left(FV^{-1}\right), \tag{11}$$

where the matrices $F = [\partial\mathscr{F}_i(x)/\partial x_j]_{x=x_0}$ and $V = [\partial\mathscr{V}_i(x)/\partial x_j]_{x=x_0}$. $\mathscr{F}_i(x)$, where x is the set of all disease free states in the compartment i, is the rate of appearance of new infections in compartment i, and $V_i(x)$ is net transfer rate (other than infections) of compartment i. The net transfer rate is given by $\mathscr{V}_i = \mathscr{V}_i^- - \mathscr{V}_i^+$, where $\mathscr{V}_i^-$ is the rate of transfer of individuals out of compartment i, and $\mathscr{V}_i^+$ is the rate of transfer of individuals into compartment i by all other means. Therefore, the disease transmission model consists of nonnegative initial conditions, $x_i(0)$, together with the following system of equations:

$$x_j' = f_j(x) \equiv \mathscr{F}_j(x) - \mathscr{V}_j, \quad j \geq 1. \tag{12}$$

From model (6), we have

$$\begin{aligned} F &= \begin{pmatrix} \dfrac{\partial\mathscr{F}_1}{\partial i(t)} & \dfrac{\partial\mathscr{F}_1}{\partial r(t)} \\ \dfrac{\partial\mathscr{F}_2}{\partial i(t)} & \dfrac{\partial\mathscr{F}_2}{\partial r(t)} \end{pmatrix} = \begin{pmatrix} \beta s & 0 \\ 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} \dfrac{\partial\mathscr{V}_1}{\partial i(t)} & \dfrac{\partial\mathscr{V}_1}{\partial r(t)} \\ \dfrac{\partial\mathscr{V}_2}{\partial i(t)} & \dfrac{\partial\mathscr{V}_2}{\partial r(t)} \end{pmatrix} = \begin{pmatrix} \theta + m + \mu & 0 \\ -\theta & \mu + \delta \end{pmatrix}. \end{aligned} \tag{13}$$

Since we have only two distinct stages, namely, $I(t)$ and $R(t)$, it follows that both F and V are 2×2 square matrices. Further, it can be noticed that F is nonnegative and V is nonsingular. The basic reproductive number $\mathscr{R}_0$ is the

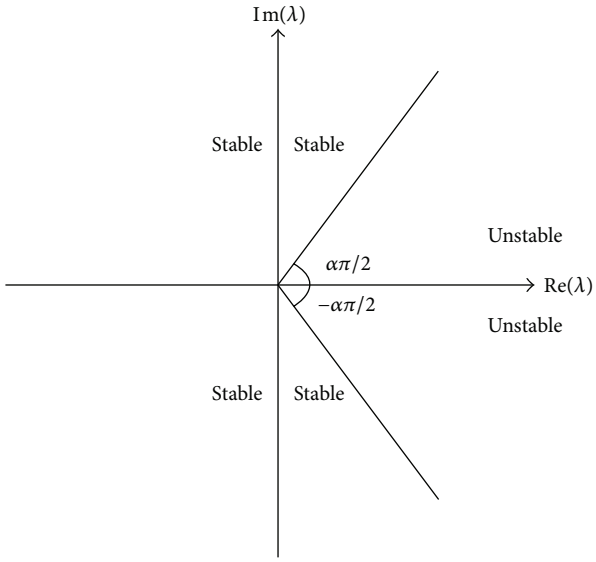

FIGURE 2: Stability region of the fractional order system with $\alpha \in (0, 1]$.

dominant eigenvalue of the matrix FV^{-1}, which is obtained by solving the characteristic equation $(FV^{-1})I - \Lambda I = 0$, where Λ is the eigenvalue and $I(t)$ is the identity matrix. At the disease-free equilibrium $\mathscr{E}_0 = (1, 0, 0, 0)$, we have

$$\mathscr{R}_0 = \frac{\beta}{\theta + m + \mu}. \tag{14}$$

The following theorem states that $\mathscr{R}_0$ is a threshold parameter for the stability of model (6).

Theorem 3. *The disease-free equilibrium is locally asymptotically stable and the infection will die out if $\mathscr{R}_0 < 1$ and is unstable if $\mathscr{R}_0 > 1$.*

Proof. The disease-free equilibrium is locally asymptotically stable if all the eigenvalues, λ_i,$i = 1, 2, 3, 4$, of Jacobian matrix $J(\mathscr{E}_0)$ satisfy condition [30]:

$$\left|\arg(\lambda_i)\right| > \frac{\alpha\pi}{2}, \tag{15}$$

where

$$J(\mathscr{E}_0) = \begin{pmatrix} -\mu & -\beta & 0 & \eta \\ 0 & \beta - (\theta + m + \mu) & 0 & 0 \\ 0 & 0 & -(\mu + \delta) & 0 \\ 0 & 0 & \delta & -(\eta + \mu) \end{pmatrix}. \tag{16}$$

Figure 2 depicts the stability region of the fractional order system, according to condition (15). The eigenvalues of Jacobian matrix $J(\mathscr{E}_0)$ are $\lambda_1 = -\mu$, $\lambda_2 = \beta - (\theta + m + \mu)$, $\lambda_3 = -(\mu + \delta)$, $\lambda_4 = -(\eta + \mu)$. Hence, $\mathscr{E}_0$ is locally asymptotically stable if $\mathscr{R}_0 < 1$ and is unstable if $\mathscr{R}_0 > 1$.

Now, we extend the analysis to endemic equilibrium $\mathscr{E}_+$. Jacobian matrix $J(\mathscr{E}_+)$ evaluated at the endemic equilibrium is

$$J(\mathscr{E}_+) = \begin{pmatrix} -\beta i^* - \mu & -\beta s^* & 0 & \eta \\ \beta i^* & \beta s^* + \sigma\beta c^* - (\theta + m + \mu) & 0 & \sigma\beta i^* \\ 0 & (1 - \sigma)\beta c^* + \theta & -(\mu + \delta) & (1 - \sigma)\beta i^* \\ 0 & -\beta c^* & \delta & -\beta i^* - (\eta + \mu) \end{pmatrix}, \tag{17}$$

with characteristic equation

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0, \tag{18}$$

where

$$\begin{aligned} a_1 &= (D_1 + D_3 + D_5), \\ a_2 &= \left(D_1D_3 - D_4\delta + D_1D_5 + D_3D_5 + \beta^2 i^* s^* + \sigma\beta^2 c^* i^*\right), \\ a_3 &= \left(D_1D_3D_5 - D_1D_4\delta + D_3\beta^2 i^* s^* + D_5\beta^2 i^* s^* + \beta^2 c^* \eta i^* \right. \\ &\quad \left. - D_2\sigma\beta\delta i^* + \sigma\beta^2 D_1 c^* i^* + \sigma D_3\beta^2 c^* i^*\right), \\ a_4 &= D_3D_5\beta^2 i^* s^* - D_2\beta\delta\eta i^* + D_3\beta^2 c^* \eta i^* \\ &\quad - D_4\beta^2\delta i^* s^* - \sigma\beta\delta D_1D_2 i^* + \sigma D_1D_3\beta^2 c^* i^*, \\ D_1 &= \beta i + \mu, \qquad D_2 = (1 - \sigma)\beta c^* + \theta, \\ D_3 &= (\mu + \delta), \qquad D_4 = (1 - \sigma)\beta i^*, \\ D_5 &= \beta i^* + (\eta + \mu). \end{aligned} \tag{19}$$

If $D(\Phi)$ denotes the discriminant of the polynomial: $\Phi(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4$, then denote

$$D(\Phi) = \begin{vmatrix} 1 & a_1 & a_2 & a_3 & a_4 & 0 & 0 \\ 0 & 1 & a_1 & a_2 & a_3 & a_4 & 0 \\ 0 & 0 & 1 & a_1 & a_2 & a_3 & a_4 \\ 4 & 3a_1 & 2a_2 & a_3 & 0 & 0 & 0 \\ 0 & 4 & 3a_1 & 2a_2 & a_3 & 0 & 0 \\ 0 & 0 & 4 & 3a_1 & 2a_2 & a_3 & 0 \\ 0 & 0 & 0 & 4 & 3a_1 & 2a_2 & a_3 \end{vmatrix}. \tag{20}$$

From [31], we have the proposition.

Proposition 4. *Assume that $\mathscr{E}_+$ exists in $\mathbf{R}^4_+$.*

(1) *Let c_1, c_2, c_3 be the Routh-Hurwitz determinants: $c_1 = a_1$, $c_2 = \begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix}$, $c_3 = \begin{vmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & a_4 & a_3 \end{vmatrix}$. Therefore, when $\alpha = 1$,*

the equilibrium point $\mathscr{E}_+$ is locally asymptotically stable if

$$c_1 > 0, \qquad c_2 > 0, \qquad c_3 = 0, \qquad a_4 > 0. \tag{21}$$

However, conditions (21) *are sufficient (not necessary) conditions for $\mathscr{E}_+$ to be locally asymptotically stable for all $\alpha \in [0, 1)$.*

(2) *If $D(\Phi) > 0$, $a_1 > 0$, $a_2 < 0$, and $\alpha > 2/3$, then the equilibrium point $\mathscr{E}_+$ is unstable.*

(3) *If $D(\Phi) < 0$, $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_4 > 0$, and $\alpha < 1/3$, then the equilibrium $\mathscr{E}_+$ is locally asymptotically stable. Also, if $D(\Phi) < 0$, $a_1 < 0$, $a_2 > 0$, $a_3 < 0$, $a_4 > 0$, then the equilibrium point $\mathscr{E}_+$ is unstable.*

(4) *If $D(\Phi) < 0$, $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_4 > 0$, and $a_2 = (a_1 a_4/a_3) + (a_3/a_1)$, then the equilibrium point $\mathscr{E}_+$ is locally asymptotically stable for all $\alpha \in (0, 1)$.*

(5) *$a_4 > 0$ is the necessary condition for the equilibrium point $\mathscr{E}_+$ to be locally asymptotically stable.* □

3. Implicit Euler's Scheme for FODEs

Since most of the FODEs do not have exact analytic solutions, so approximation and numerical techniques must be used. In addition, most of resulting biological systems are stiff (one definition of the stiffness is that the global accuracy of the numerical solution is determined by stability rather than local error and implicit methods are more appropriate for it). The *stiffness* often appears due to the differences in speed between the fastest and slowest components of the solutions and stability constraints. In addition, the state variables of these types of models are very sensitive to small perturbations (or changes) in the parameters which occur in the model. Therefore, efficient use of a reliable numerical method for dealing with stiff problems is necessary.

Consider the following fractional order differential equation:

$$\begin{aligned} &D^{\alpha} y(t) = f\left(t, y(t)\right), \quad t \in [0, T], \\ &\qquad\qquad\qquad\qquad 0 < \alpha \le 1. \\ &y^{(k)}(0) = y^{(k)}(0), \quad k = 0, 1, 2, \ldots, m-1, \end{aligned} \tag{22}$$

Here, $y(t) = [y_1(t), y_2(t), \ldots, y_n(t)]^T$ and $f(t, y(t))$ satisfy the Lipschitz condition in variable y:

$$\left\| f\left(t, y(t)\right) - f\left(t, x(t)\right) \right\| \le K \left\| y(t) - x(t) \right\|, \quad K > 0, \tag{23}$$

where $x(t)$ is the solution of the perturbed system.

Theorem 5. *Problem* (22) *has a unique solution provided that Lipschitz condition* (23) *is satisfied and $\overline{M} = KT^{\alpha}/\Gamma(\alpha + 1) < 1$.*

Proof. Using the definitions of Section 1, we can apply a fractional integral operator to the differential equation (22) and incorporate the initial conditions, thus converting the equation into the equivalent equation:

$$y(t) = \sum_{k=0}^{m-1} y_0^{(k)} \frac{t^k}{k!} + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f\left(s, y(s)\right) ds, \tag{24}$$

which also is a Volterra equation of the second kind. Define operator $\mathscr{L}$, such that

$$\mathscr{L} y(t) = \sum_{k=0}^{m-1} y_0^{(k)} \frac{t^k}{k!} + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f\left(s, y(s)\right) ds. \tag{25}$$

Then, we have

$$\begin{aligned} &\left\| \mathscr{L} y(t) - \mathscr{L} x(t) \right\| \\ &\quad \le \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \left\| f\left(s, y(s)\right) - f\left(s, x(s)\right) \right\| ds \\ &\quad \le \frac{K}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \sup_{s \in [0,T]} \left| y(s) - x(s) \right| ds \\ &\quad \le \frac{K}{\Gamma(\alpha)} \left\| y - x \right\| \int_0^t s^{\alpha-1} ds \\ &\quad \le \frac{KT^{\alpha}}{\Gamma(\alpha+1)} \left\| y - x \right\| T^{\alpha}. \end{aligned} \tag{26}$$

So, we obtain

$$\left\| \mathscr{L} y(t) - \mathscr{L} x(t) \right\| \le \overline{M} \left\| y - x \right\|. \tag{27}$$

By Banach contraction principle [32], we can deduce that $\mathscr{L}$ has a unique fixed point which implies that our problem has a unique solution. □

Several numerical methods have been proposed to solve the FODEs [11, 33]. Recently, the predictor-corrector algorithm is an efficient and powerful technique for solving the FODEs, which is a generalization of the Adams-Bashforth-Moulton method. The modification of Adams-Bashforth-Moulton algorithm is proposed by Diethelm [34, 35] to approximate the fractional order derivative. However, converted Volterra integral equation (24) is with a weakly singular kernel, such that a regularization is not necessary any more. It seems that there exist only a very small number of software packages for nonlinear Volterra equations. In our case, the kernel may not be continuous, and therefore the classical numerical algorithms for the integral part of (24) are unable to handle the solution of (22). Therefore, we implement the implicit Euler's scheme to approximate the fractional order derivative.

Given model (22) and mesh points $\mathscr{T} = \{t_0, t_1, \ldots, t_N\}$, such that $t_0 = 0$ and $t_N = T$, then a discrete approximation to the fractional derivative can be obtained by a simple quadrature formula, using Caputo's fractional derivative (3)

of order $\alpha, 0 < \alpha \leq 1$ and using *implicit* Euler's approximation as follows (see [12]):

$$
\begin{aligned}
D_*^{\alpha} x_i(t_n) &= \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{dx_i(s)}{ds} (t_n - s)^{-\alpha} ds \\
&\approx \frac{1}{\Gamma(1-\alpha)} \\
&\quad \times \sum_{j=1}^{n} \int_{(j-1)h}^{jh} \left[\frac{x_i^j - x_i^{j-1}}{h} + O(h) \right] (nh - s)^{-\alpha} ds \\
&= \frac{1}{(1-\alpha)\Gamma(1-\alpha)} \\
&\quad \times \sum_{j=1}^{n} \left\{ \left[\frac{x_i^j - x_i^{j-1}}{h} + O(h) \right] \right. \\
&\qquad \left. \times \left[(n-j+1)^{1-\alpha} - (n-j)^{1-\alpha} \right] \right\} h^{1-\alpha} \\
&= \frac{1}{(1-\alpha)\Gamma(1-\alpha)} \frac{1}{h^{\alpha}} \\
&\quad \times \sum_{j=1}^{n} \left[x_i^j - x_i^{j-1} \right] \left[(n-j+1)^{1-\alpha} - (n-j)^{1-\alpha} \right] \\
&\quad + \frac{1}{(1-\alpha)\Gamma(1-\alpha)} \\
&\quad \times \sum_{j=1}^{n} \left[x_i^j - x_i^{j-1} \right] \\
&\qquad \times \left[(n-j+1)^{1-\alpha} - (n-j)^{1-\alpha} \right] O\left(h^{2-\alpha}\right).
\end{aligned}
\tag{28}
$$

Setting

$$
\begin{aligned}
\mathcal{G}(\alpha, h) &= \frac{1}{(1-\alpha)\Gamma(1-\alpha)} \frac{1}{h^{\alpha}}, \\
\omega_j^{\alpha} &= j^{1-\alpha} - (j-1)^{1-\alpha}, \quad (\text{where } \omega_1^{\alpha} = 1),
\end{aligned}
\tag{29}
$$

then the first-order approximation method for the computation of Caputo's fractional derivative is then given by the expression

$$
D_*^{\alpha} x_i(t_n) = \mathcal{G}(\alpha, h) \sum_{j=1}^{n} \omega_j^{\alpha} \left(x_i^{n-j+1} - x_i^{n-j} \right) + O(h). \tag{30}
$$

From the analysis and numerical approximation, we also arrive at the following proposition.

Proposition 6. *The presence of a fractional differential order in a differential equation can lead to a notable increase in the complexity of the observed behavior, and the solution continuously depends on all the previous states.*

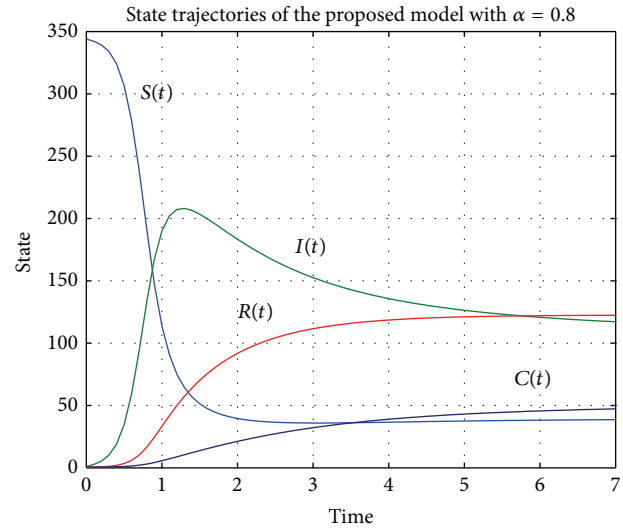

FIGURE 3: Numerical simulation of fractional order epidemic model (6), when $\alpha = 0.8$, and $\mathcal{R}_0 > 1$ (each infected individual infects more than one other member of the population and a self-sustaining group of infectious individuals will propagate), with parameter values of Table 1.

3.1. Stability and Convergence. We here prove that fractional order implicit difference approximation (30) is unconditionally stable. It follows then that the numerical solution converges to the exact solution as $h \to 0$. In order to study the stability of the numerical method, let us consider a test problem of linear scaler fractional differential equation

$$
D_*^{\alpha} u(t) = \rho_0 u(t) + \rho_1, \qquad U(0) = U_0 \tag{31}
$$

such that $0 < \alpha \leq 1$, and $\rho_0 < 0$, $\rho_1 > 0$ are constants.

Theorem 7. *The fully implicit numerical approximation* (30)*, to test problem* (31) *for all $t \geq 0$, is consistent and unconditionally stable.*

Proof. We assume that the approximate solution of (31) is of the form $u(t_n) \approx U^n \equiv \zeta_n$; then (31) can be reduced to

$$
\begin{aligned}
&\left(1 - \frac{\rho_0}{\mathcal{G}_{\alpha,h}}\right) \zeta_n \\
&\quad = \zeta_{n-1} + \sum_{j=2}^{n} \omega_j^{(\alpha)} \left(\zeta_{n-j} - \zeta_{n-j+1} \right) + \frac{\rho_1}{\mathcal{G}_{\alpha,h}}, \quad n \geq 2,
\end{aligned}
\tag{32}
$$

or

$$
\zeta_n = \frac{\zeta_{n-1} + \sum_{j=2}^{n} \omega_j^{(\alpha)} \left(\zeta_{n-j} - \zeta_{n-j+1} \right) + \rho_1 / \mathcal{G}_{\alpha,h}}{\left(1 - \left(\rho_0 / \mathcal{G}_{\alpha,h}\right)\right)}, \quad n \geq 2. \tag{33}
$$

Since $(1 - (\rho_0/\mathcal{G}_{\alpha,h})) \geq 1$, for all $\mathcal{G}_{\alpha,h}$, then

$$
\zeta_1 \leq \zeta_0, \tag{34}
$$

$$
\zeta_n \leq \zeta_{n-1} + \sum_{j=2}^{n} \omega_j^{(\alpha)} \left(\zeta_{n-j} - \zeta_{n-j+1} \right), \quad n \geq 2. \tag{35}
$$

Table 1: List of parameters.

Parameter	Description	Value	Reference
μ	Replacement and exit rate (day^{-1})	0.011	[36]
β	Contact (transmission) rate of suspectable to be infected (animal^{-1} day^{-1})	0.15	[36]
θ	Recovery rate of infected animals day $^{-1}$	0.16	Assumed
m	Disease-induced mortality rate (day^{-1})	0.041	Assumed
η	Cross-immune period	0.5	[36]
σ	The average reinfection probability of $C(t)$	0.06	Assumed
δ	The average time of appearance of new dominant clusters	1	Assumed
N	The total number of population	345	Assumed

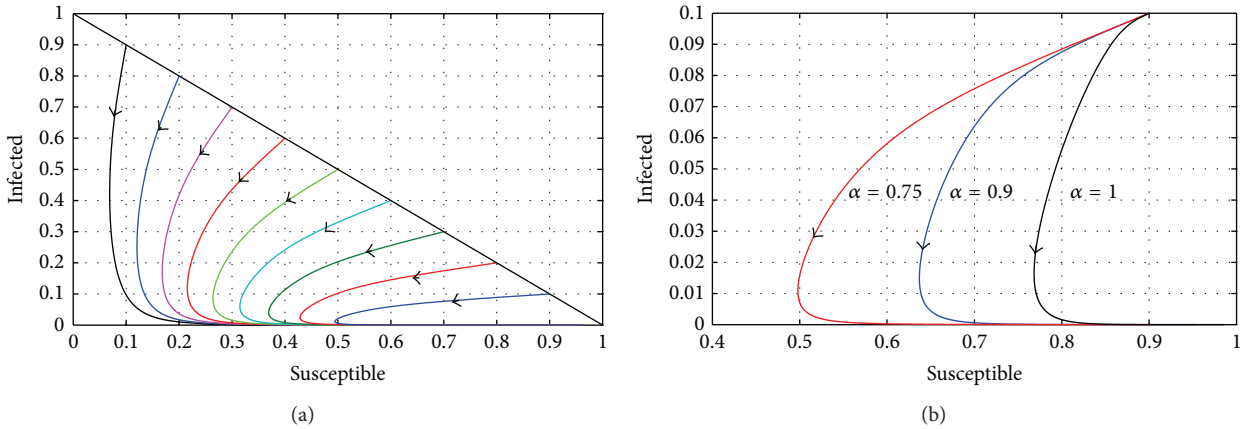

Figure 4: Phase plane portrait for fractional order endemic model (6), in absence of $C(t)$ and $R(t)$ components, when $\alpha = 0.7$ (a) and $\alpha = 0.9$ (b) with $\mathscr{R}_0 = 0.5 < 1$. We note that solution paths approach the disease-free equilibrium $\mathscr{E}_0 = (1, 0, 0)$.

Thus, for $n = 2$, the above inequality implies

$$\zeta_2 \leq \zeta_1 + \omega_2^{(\alpha)} (\zeta_0 - \zeta_1). \tag{36}$$

Using relation (34) and the positivity of the coefficients ω_2, we get

$$\zeta_2 \leq \zeta_1. \tag{37}$$

Repeating the process, we have, from (35),

$$\zeta_n \leq \zeta_{n-1} + \sum_{j=2}^{n} \omega_j^{(\alpha)} \left(\zeta_{n-j} - \zeta_{n-j+1}\right) \leq \zeta_{n-1}, \tag{38}$$

since each term in the summation is negative. Thus, $\zeta_n \leq \zeta_{n-1} \leq \zeta_{n-2} \leq \cdots \leq \zeta_0$. With the assumption that $\zeta_n = |U^n| \leq \zeta_0 = |U^0|$, which entails $\|U^n\| \leq \|U_0\|$, we have stability. □

Of course this numerical technique can be used both for linear and for nonlinear problems, and it may be extended to multiterm FODEs.

3.2. Numerical Simulations. The approximate solutions of epidemic model (6) are displayed in Figures 3, 4, and 5, and sensitivity of $\mathscr{R}_0$ to transmission coefficients is displayed in Figure 6. The numerical simulations are performed by Euler's implicit scheme. We choose commensurate fractional order that $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_4 = \alpha$, with different fractional order values and the parameter values given in Table 1.

4. Conclusions

In this paper, we provided a fractional order SIRC epidemic model with *Salmonella* bacteria infection. We derived the sufficient conditions to preserve the asymptotic stability of infection-free and endemic steady states. The threshold parameter (reproduction number) $\mathscr{R}_0$ has been evaluated in terms of contact rate, recovery rate, and other parameters in the model. We provided unconditionally stable method, using Euler's implicit method for the fractional order differential system. The solution of a fractional order model at any time t^* continuously depends on all the previous states at $t \leq t^*$. Fractional order dynamical models are more suitable to model biological systems with memory than their integer-orders. The presence of a fractional differential order into a corresponding differential equation leads to a notable increase in the complexity of the observed behavior and enlarges the stability region of the solutions. However, fractional order differential models have the same integer-order counterpart steady states, when $\alpha > 0.5$.

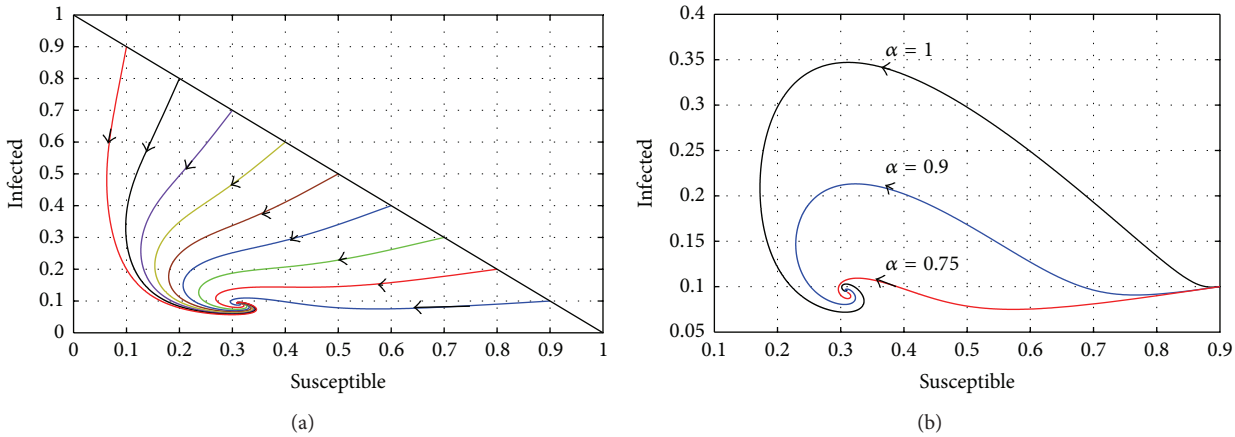

FIGURE 5: Phase plane portrait for classic fractional order endemic model (6) when $\alpha = 1$ (a) and $\alpha = 0.9$ (b) with $\mathscr{R}_0 = 1.2 > 1$. We note that solution paths approach the endemic equilibrium $\mathscr{E}_+$ given by (7).

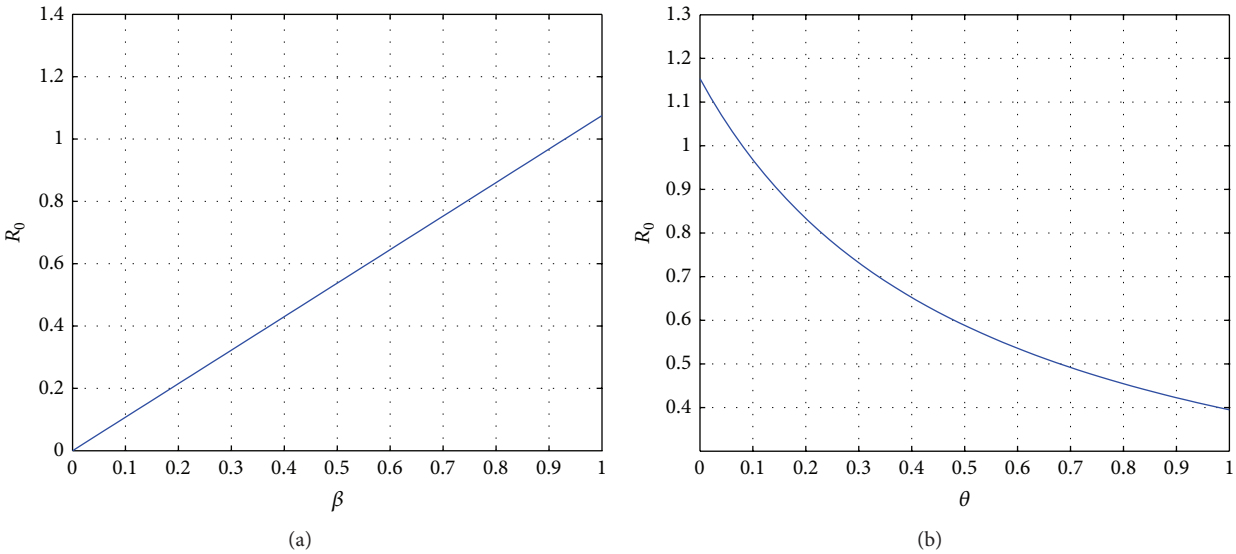

FIGURE 6: Sensitivity of $\mathscr{R}_0$ with respect to the transmission coefficients β and θ.

Conflict of Interests

The authors declare that they have no competing interests in this paper.

Acknowledgments

This work was supported by UAE University (UAEU-NRF-7-20886). The authors would like to thank Dr. Ali Bhrawy for his valuable comments.

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