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Abstract

Infectious disease has an influence on the density of a population. In this paper, a fractional-order logistic growth model with infectious disease is formulated. The population grows logistically and divided into two compartments i.e. susceptible and infected populations. We start by investigating the existence, uniqueness, non-negativity, and boundedness of solutions. Furthermore, we show that the model has three equilibrium points namely the population extinction point, the disease-free point, and the endemic point. The population extinction point is always a saddle point while others are conditionally asymptotically stable. For the non-trivial equilibrium points, we successfully show that the local and global asymptotic stability have the similar properties. Especially, when the endemic point exists, it is always globally asymptotically stable. We also show the existence of forward bifurcation in our model. We portray some numerical simulations consist of the phase portraits, time series, and a bifurcation diagram to validate the analytical findings.

Keywords: Dynamics, Fractional-Order, Logistic Growth, Infectious Disease

1. Introduction

The logistic growth model is first proposed by a Belgian mathematician called Pierre François Verhulst [1], which given by a first-order differential equation

$$\frac{dN}{dt} = rN \left(1 - \frac{N}{K} \right), \tag{1}$$

where r is the intrinsic growth rate and K is the environmental carrying capacity of the population. The logistic growth model is considered more realistic rather than the exponential growth model proposed by Malthus [2]. To achieve a more specific condition of natural phenomena, the logistic growth model is modified such as Richards model [3], Blumberg model [4], and Tsoularis model [5]. Some modifications are also done to facilitate the biological behavior of populations for instance: the Allee effect [6–8] and feedback control [9, 10]. It is also applied in interaction between populations in predator-prey schemes as in [11–16]. Nowadays, the logistic model and its modifications are used to modeling and predicting the infected population of new coronavirus 2019 (COVID-19), see [17–21].

In this paper, we modify the logistic growth model (1) by assuming the population is infected by a disease. We divide the population $N(t)$ into two compartments namely susceptible population $S(t)$ and infected population $I(t)$. We also assume that the infection rate is bilinear and the infected population may recover by its immune system. This model is given by a first-order differential equations as follows.

$$\begin{aligned}
\frac{dS}{dt} &= rS \left(1 - \frac{S+I}{K} \right) - \beta SI + \omega I, \\
\frac{dI}{dt} &= \beta SI - (\omega + \delta)I,
\end{aligned} \tag{2}$$

where β is the infection rate, ω is the recovery rate, and δ is the death rate causes by disease. It is clear that the total population $N(t)$ satisfies $N(t) = S(t) + I(t)$. Some similar works are done by scholars [10, 22–29]. The novelty of our model compared with their models lies on the utilization of logistic growth rate rather than the constant growth rate.

To attain more realistic model, we apply the fractional-order derivative as the operator. The model with fractional-order derivative describe a preferable biological condition rather than the first-order derivative since

this operator has capability in involving all previous condition to express the state condition which called the memory effect, see [30–35]. By replacing the first-order derivative at the left hand side of model (2) with the fractional-order derivative and redefine some parameters in similar way with [32] to obtain the same dimension of (time)$^\alpha$, we get

$$
\begin{aligned}
{}^C\mathcal{D}_t^\alpha S &= rS\left(1 - \frac{S+I}{K}\right) - \beta SI + \omega I = G_1(N), \\
{}^C\mathcal{D}_t^\alpha I &= \beta SI - (\omega + \delta)I = G_2(N),
\end{aligned}
\tag{3}
$$

where ${}^C\mathcal{D}_t^\alpha$ is Caputo fractional-order derivative define by

$$
{}^C\mathcal{D}_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} f'(\tau) d\tau,
\tag{4}
$$

with $t \geq 0$, $f \in C^n([0, +\infty), \mathbb{R})$, and $\Gamma(\cdot)$ is a Gamma function [36].

This paper is organized as follows. To ensure the model (3) meets the expected biological conditions, the existence, uniqueness, non-negativity, and boundedness are given in Sections 2 and 3. In Section 4, we study the dynamics of model (3) including the existence of equilibrium points, their local and global stability, and the existence of forward bifurcation. Finally, we close our works by giving some concluding remarks in Section 5.

2. Existence and Uniqueness

Suppose that $\Theta := \left\{ (S, I) \in \mathbb{R}^2 : \max(|S|, |I|) \leq \sigma \right\}$. We will show that for each initial values in Θ, the model (3) has a unique solution in the region $\Theta \times (0, T]$, $T < \infty$. The similar approach used in [31, 32] is employed. We consider a mapping $G(N) = (G_1(N), G_2(N))$. For any $N = (S, I)$, $\bar{N} = (\bar{S}, \bar{I})$, $N, \hat{N} \in \Theta$, we acquire

$$
\begin{aligned}
\|G(N) - G(\bar{N})\| &= |G_1(N) - G_1(\bar{N})| + |G_2(N) - G_2(\bar{N})| \\
&= \left| \left(rS\left(1 - \frac{S+I}{K}\right) - \beta SI + \omega I \right) - \left(r\bar{S}\left(1 - \frac{\bar{S}+\bar{I}}{K}\right) - \beta \bar{S}\bar{I} + \omega \bar{I} \right) \right| + \\
&\quad \left| (\beta SI - (\omega + \delta)I) - (\beta \bar{S}\bar{I} - (\omega + \delta)\bar{I}) \right| \\
&= \left| r(S - \bar{S}) - \frac{r}{K}(S^2 - \bar{S}^2) - \left(\frac{r}{K} + \beta\right)(SI - \bar{S}\bar{I}) + \omega(I - \bar{I}) \right| + \\
&\quad \left| \beta(SI - \bar{S}\bar{I}) - (\omega + \delta)(I - \bar{I}) \right| \\
&\leq r|S - \bar{S}| + \frac{r}{K}\left|S^2 - \bar{S}^2\right| + \left(\frac{r}{K} + \beta\right)|SI - \bar{S}\bar{I}| + \omega|I - \bar{I}| + \\
&\quad \beta|SI - \bar{S}\bar{I}| + (\omega + \delta)|I - \bar{I}| \\
&= r|S - \bar{S}| + \frac{r}{K}|S + \bar{S}||S - \bar{S}| + \left(\frac{r}{K} + 2\beta\right)|I(S - \bar{S}) + \bar{S}(I - \bar{I})| + (2\omega + \delta)|I - \bar{I}| \\
&\leq r|S - \bar{S}| + \frac{2\sigma r}{K}|S - \bar{S}| + \left(\frac{r}{K} + 2\beta\right)\sigma|S - \bar{S}| + \left(\frac{r}{K} + 2\beta\right)\sigma|I - \bar{I}| + (2\omega + \delta)|I - \bar{I}| \\
&= \left(r + \frac{3\sigma r}{K} + 2\sigma\beta\right)|S - \bar{S}| + \left(\frac{\sigma r}{K} + 2\sigma\beta + 2\omega + \delta\right)|I - \bar{I}| \\
&\leq L\|N - \bar{N}\|,
\end{aligned}
$$

where $L = \max\left\{ r + \dfrac{3\sigma r}{K} + 2\sigma\beta, \dfrac{\sigma r}{K} + 2\sigma\beta + 2\omega + \delta \right\}$, and hence $G(N)$ satisfies the Lipschitz condition. Obeying Theorem 3.7 in [37], the existence and uniqueness of solution of model (3) is satisfied. Therefore, the following theorem is preserved.

Theorem 1. *For each non-negative initial condition in Θ, there exists a unique solution of model (3) in the region $\Theta \times (0, T]$.*

3. Non-negativity and Boundedness

The non-negativity and boundedness of solutions of model (3) are given by the following theorem.

Theorem 2. *For each non-negative initial conditions, the solutions of model (3) are always non-negative and uniformly bounded.*

proof. By using conformable manner as in [31] and applying Lemma 2 in [32], it can be clarified that the solutions are non-negative if the initial conditions are also non-negative. Furthermore, we give the proof of the boundedness of solutions. We confirm that the total population $N(t)$ satisfies $N(t) = S(t) + I(t)$, and hence we get

$$
\begin{aligned}
{}^{C}\mathcal{D}_t^\alpha N(t) + \delta N(t) &= rS\left(1 - \frac{S+I}{K}\right) - \beta SI + \omega I + \beta SI - (\omega + \delta)I + \delta S + \delta I \\
&= (r+\delta)S - \frac{rS^2}{K} - \frac{rSI}{K} \\
&= -\frac{r}{K}\left(S - \frac{(r+\delta)K}{2r}\right)^2 + \frac{(r+\delta)^2 K}{4r} - \frac{rSI}{K} \\
&\leq \frac{(r+\delta)^2 K}{4r}.
\end{aligned}
$$

Based on Lemma 3 in [38], we have

$$
N(t) \leq \left(N(0) - \frac{(r+\delta)^2 K}{4r}\right) E_\alpha\left[-\delta t^\alpha\right] + \frac{(r+\delta)^2 K}{4r},
$$

where E_α is mittag-leffler function. Therefore, we have $N(t) \leq \frac{(r+\delta)^2 K}{4r}$ for $t \to \infty$, which convince all solutions are confined to the region Ψ where

$$
\Psi := \left\{(x,y) \in \mathbb{R}_+^2 \ : \ S + I \leq \frac{(r+\delta)^2 K}{4r} + \varepsilon, \ \varepsilon > 0\right\}.
$$

Consequently, all solutions of model (3) are uniformly bounded. ∎

4. Equilibrium Points and Their Stability

We acquire the equilibrium point by solving $G_1(N) = G_2(N) = 0$. In consequence, we obtain three equilibrium points i.e the population extinction point $\Omega_0 = (0,0)$, the disease-free point $\Omega_1 = (K,0)$, and the endemic equilibrium point $\Omega_2 = \left(\frac{K}{R_0}, \left(1 - \frac{1}{R_0}\right)\frac{rK}{r+\delta R_0}\right)$ where $R_0 = \frac{\beta K}{\omega + \delta}$. Furthermore, the dynamics of model (3) consist of the local and global stability are shown by the following theorems.

Theorem 3. *The population extinction point $\Omega_0 = (0,0)$ is always a saddle point.*

proof. We compute the Jacobian matrix of model (3) at Ω_0. Thus, we have

$$
J(\Omega_0) = \begin{bmatrix} r & \omega \\ 0 & -(\omega + \delta) \end{bmatrix}, \tag{5}
$$

which gives the eigenvalues: $\lambda_1 = r$ and $\lambda_2 = -(\omega + \delta)$. We confirm that $|\arg(\lambda_1)| < \alpha\pi/2$ and $|\arg(\lambda_2)| > \alpha\pi/2$, which ensures that Ω_0 is always a saddle point, see Theorem 3 in [32]. ∎

Theorem 4. *If $R_0 < 1$ then the disease-free point $\Omega_1 = (K,0)$ is asymptotically stable both locally and globally. Otherwise, it is a saddle point.*

proof. We start by identify the local stability of Ω_1. For $\Omega_1 = (K,0)$, the Jacobian matrix of model (3) is

$$
J(\Omega_1) = \begin{bmatrix} -r & -\left(\frac{r}{K} + \beta\right)K + \omega \\ 0 & (\omega + \delta)(R_0 - 1) \end{bmatrix}. \tag{6}
$$

From (6), we have the eigenvalues: $\lambda_1 = -r$ which gives $|\arg(\lambda_1)| > \alpha\pi/2$ and $\lambda_2 = (\omega + \delta)(R_0 - 1)$ where $|\arg(\lambda_2)|$ depends on the value of R_0. If $R_0 < 1$ then $|\arg(\lambda_2)| > \alpha\pi/2$, and If $R_0 > 1$ then $|\arg(\lambda_2)| < \alpha\pi/2$. By applying Theorem 3 in [32], the local dynamics of model (3) is emphasized.

Now, we give the proof that the global stability of Ω_1 has the similar properties as its local one. We define a positive Lyapunov function as follows.

$$\Phi_1(S, I) = \left[S - K - K \ln \frac{S}{K} \right] + \frac{(r + \beta K) R_0}{\beta K} I. \tag{7}$$

According to Lemma 3.1 in [39], the fractional-order derivative of (7) is given by

$$
\begin{aligned}
{}^C \mathcal{D}_t^\alpha \Phi_1(S, I) &\leq \left(\frac{S - K}{S} \right) {}^C \mathcal{D}_t^\alpha S + \frac{(r + \beta K) R_0}{\beta K} {}^C \mathcal{D}_t^\alpha I \\
&= \left(\frac{S - K}{S} \right) \left(rS \left(1 - \frac{S + I}{K} \right) - \beta SI + \omega I \right) + \frac{(r + \beta K) R_0}{\beta K} \left(\beta SI - (\omega + \delta) I \right) \\
&= (S - K) \left(r - \frac{rS + rI}{K} - \beta I + \omega \frac{I}{S} \right) + \frac{(r + \beta K) R_0 SI}{K} - \frac{(\omega + \delta)(r + \beta K) R_0}{\beta K} I \\
&= \left(2rS - rK - \frac{rS^2}{K} - \frac{rSI}{K} + rI - \beta SI + \beta KI + \omega I - \frac{\omega KI}{S} \right) + \frac{(r + \beta K) R_0 SI}{K} - (r + \beta K) I \\
&= -\frac{rS^2}{K} + 2rS - rK - \left(\frac{r}{K} + \beta - \frac{(r + \beta K) R_0}{K} \right) SI - \frac{\omega KI}{S} \\
&= -\frac{r}{K} (S - K)^2 - \frac{\omega KI}{S} - \frac{(1 - R_0)(r + \beta K) SI}{K}.
\end{aligned}
$$

Since $R_0 < 1$, we conclude that ${}^C \mathcal{D}_t^\alpha \Phi_1(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$. We also ensure that ${}^C \mathcal{D}_t^\alpha \Phi_1(S, I) = 0$ implies that $(S, I) = (K, 0)$. Therefore, the only invariant set on which ${}^C \mathcal{D}_t^\alpha \Phi_1(S, I) = 0$ is the singleton $\{\Omega_1\}$. Obeying Lemma 4.6 in [40], the disease-free point $\Omega_1 = (K, 0)$ is globally asymptotically stable. ∎

Remark 1. From Theorem 4, we conclude that if $R_0 < 1$ then Ω_1 is locally and globally asymptotically stable. For all initial conditions, the disease will become extinct, the density of population will eventually increases, and tends to the environmental carrying capacity.

Theorem 5. *The endemic equilibrium point* $\Omega_2 = \left(\dfrac{K}{R_0}, \left(1 - \dfrac{1}{R_0} \right) \dfrac{rK}{r + \delta R_0} \right)$ *is always asymptotically stable both locally and globally.*

proof. Suppose that

$$
\begin{aligned}
\xi_1 &= \frac{R_0 \xi_2}{\beta K} \left[1 - \frac{r + (\omega + \delta) R_0}{r + \delta R_0} \right] - \frac{r}{R_0}, \\
\xi_2 &= \left(1 - \frac{1}{R_0} \right) \frac{\beta rK}{R_0}.
\end{aligned}
$$

It is easy to verify that $\xi_1 < 0$ and $\xi_2 > 0$. By evaluating Jacobian matrix of model (3) at Ω_2, we acquire

$$J(\Omega_2) = \begin{bmatrix} \xi_1 & -\dfrac{r + \delta R_0}{R_0} \\[2ex] \dfrac{R_0 \xi_2}{r + \delta R_0} & 0 \end{bmatrix}, \tag{8}$$

and obtain a pair of eigenvalues: $\lambda_1 = \frac{\xi_1}{2} \pm \frac{1}{2} \sqrt{\xi_1^2 - 4\xi_2}$. For $\xi_1^2 < 4\xi_2$, those eigenvalues are a pair of complex conjugate. Because the real part $\frac{\xi_1}{2} < 0$, then we have $|\arg(\lambda_{1,2})| > \frac{\alpha \pi}{2}$. Therefore, Ω_2 is locally asymptotically stable. For $\xi_1^2 \geq 4\xi_2$, according to the Routh-Hurwitz theorem for Caputo fractional order [41], Ω_2 is locally asymptotically stable if $\xi_1 < 0$. Since $\xi_1 < 0$, the endemic point Ω_2 is always locally asymptotically stable. Now we will show the global stability of Ω_2 also has the similar properties with the local one. Let $S^* = \dfrac{K}{R_0}$ and $I^* = \left(1 - \dfrac{1}{R_0} \right) \dfrac{rK}{r + \delta R_0}$. Therefore, we define a positive Lyapunov function as follows.

$$\Phi_2(S, I) = \left[S - S^* - S^* \ln \frac{S}{S^*} \right] + \kappa \left[I - I^* - I^* \ln \frac{I}{I^*} \right],$$

Conforming Lemma 3.1 in [39], we obtain

$$
\begin{aligned}
{}^{C}\mathcal{D}_t^{\alpha}\Phi_2(S,I) &\leq \left(\frac{S-S^*}{S}\right){}^{C}\mathcal{D}_t^{\alpha}S + \kappa\left(\frac{I-I^*}{I}\right){}^{C}\mathcal{D}_t^{\alpha}I \\
&= \left(\frac{S-S^*}{S}\right)\left(rS\left(1-\frac{S+I}{K}\right) - \beta SI + \omega I\right) + \kappa\left(\frac{I-I^*}{I}\right)(\beta SI - (\omega+\delta)I) \\
&= (S-S^*)\left(r\left(1-\frac{S+I}{K}\right) - \beta I + \frac{\omega I}{S}\right) + \kappa(I-I^*)(\beta S - (\omega+\delta)) \\
&= (S-S^*)\left(r - \frac{rS}{K} - \frac{rI}{K} - \beta I + \frac{\omega I}{S}\right) + \kappa(I-I^*)(\beta S - (\omega+\delta)) \\
&= (S-S^*)\left(\frac{rS^*}{K} + \frac{rI^*}{K} + \beta I^* - \frac{\omega I^*}{S^*} - \frac{rS}{K} - \frac{rI}{K} - \beta I + \frac{\omega I}{S}\right) + \kappa\beta(I-I^*)(S-S^*) \\
&= (S-S^*)\left(-\frac{r}{K}(S-S^*) - \frac{r}{K}(I-I^*) - \beta(I-I^*) - \frac{\omega I^*}{S^*} + \frac{\omega I}{S}\right) + \kappa\beta(I-I^*)(S-S^*) \\
&= -\frac{r}{K}(S-S^*)^2 - \left(\frac{r}{K} + \beta - \kappa\beta\right)(S-S^*)(I-I^*) - \omega(S-S^*)\left(\frac{-S(I-I^*) + I(S-S^*)}{S^*S}\right) \\
&= -\left(\frac{r}{K} + \frac{\omega I}{S^*S}\right)(S-S^*)^2 - \left(\frac{r+\beta K}{K} - \kappa\beta - \frac{\omega}{S^*}\right)(S-S^*)(I-I^*)
\end{aligned}
$$

By choosing $\kappa = \dfrac{r+\delta R_0}{\beta K}$, we get

$$
{}^{C}\mathcal{D}_t^{\alpha}\Phi_2(S,I) \leq -\left(\frac{r}{K} + \frac{\omega I}{S^*S}\right)(S-S^*)^2.
$$

Therefore, ${}^{C}\mathcal{D}_t^{\alpha}\Phi_2(S,I) \leq 0$ for all $(S,I) \in \mathbb{R}_+^2$, and ${}^{C}\mathcal{D}_t^{\alpha}\Phi_2(S,I) = 0$ implies that $(S,I) = (S^*,I^*)$ which means the singleton $\{\Omega_2\}$ is the only invarian set on which ${}^{C}\mathcal{D}_t^{\alpha}\Phi_2(S,I) = 0$. By utilizing Lemma 4.6 in [40], the endemic point Ω_2 is always globally asymptotically stable. ∎

Remark 2. According to Theorem 5, if $R_0 > 1$, then Ω_2 is locally and globally asymptotically stable. For all positive initial conditions, the disease always exists as $t \to \infty$ and convergent to an endemic point Ω_2. Although the disease will not extinct, when $R_0 > 1$ the infected population has a bounded density.

Theorem 6. *The equilibrium point Ω_1 undergoes a forward bifurcation when R_0 passes through the critical point $R_0^* = 1$.*

proof. When $R_0 < R_0^*$, the disease-free point Ω_1 is the only non-trivial equilibrium point of model (3) which is always asymptotically stable. When $R_0 > R_0^*$, Ω_1 losses its stability and an always asymptotically stable Ω_2 occurs simultaneously. By considering $I^* = \left(1 - \dfrac{1}{R_0}\right)\dfrac{rK}{r+\delta R_0}$, we confirm that the increase of R_0 means the increase of the infected populations. ∎

Remark 3. Theorem 6 and the value of I^* in Theorem 5 show us that R_0 has an important rule in determining the existence of the infected population. If R_0 is enlarged, the intrinsic growth rate of infected populations is increased. In some literature, R_0 is called the ratio reproduction number, which is not discussed in detail in this article.

5. Numerical Simulations

Some numerical simulations are demonstrated to confirm all analytical results. The predictor-corrector scheme for Caputo fractional-order derivative developed by Diethelm [42] is applied. Since the model is studied qualitatively and there are limitations in getting the field data, we take the suppositional values as the parameters. By fixing parameter values: $r = 0.5$, $K = 5$, $\Omega = 0.2$, and $\delta = 0.1$, and varying the parameter of infection rate β in interval $0 < \beta < 0.24$ (so that $0 < R_0 < 4$) we plot the bifurcation diagram in Figure 1. The disease-free point Ω_1 which is asymptotically stable when $R_0 < 1$ is separated into two equilibrium points namely the unstable Ω_1 and the asymptotically stable endemic point Ω_2 when $R_0 > 1$. Ω_1 losses its stability when β passes through $\beta^* = 0.06$ or R_0 passes through $R_0^* = 1$, and an asymptotically stable equilibrium point appears in the interior simultaneously. Based on Theorem 6, this phenomenon is called forward bifurcation.
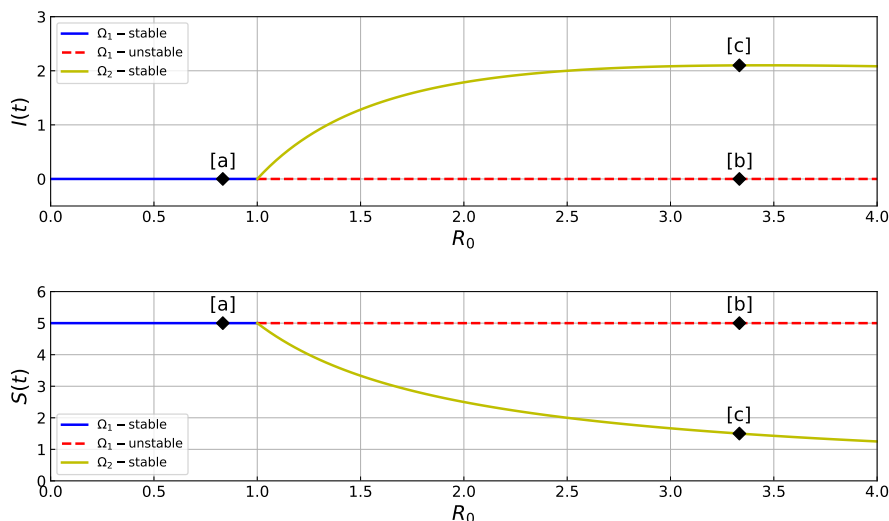
Figure 1. Bifurcation diagram of model (3) driven by β with paramater values: $r = 0.5, K = 5, \Omega = 0.2$, and $\delta = 0.1$.
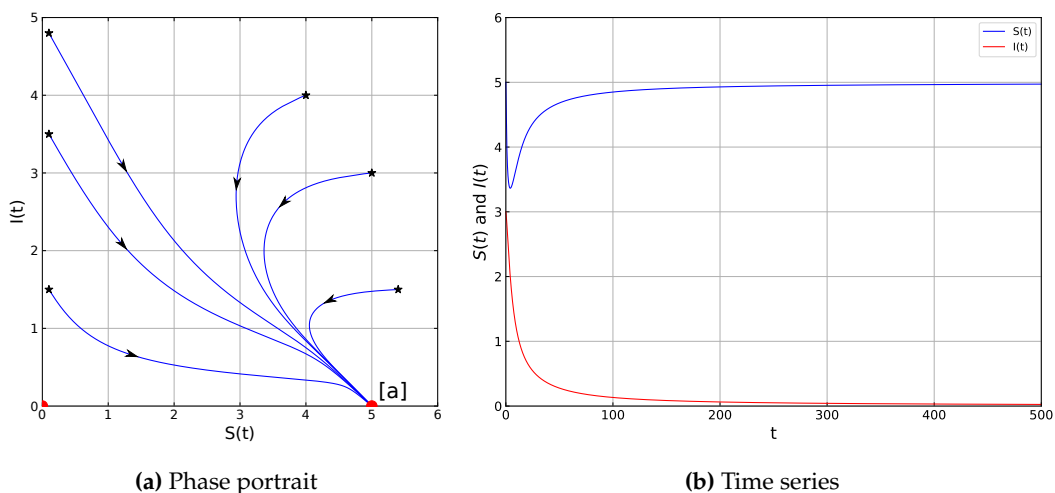

(a) Phase portrait   **(b)** Time series

Figure 2. Numerical simulation of model (3) with parameter values: $r = 0.5, K = 5, \beta = 0.05 \ \Omega = 0.2$, and $\delta = 0.1$

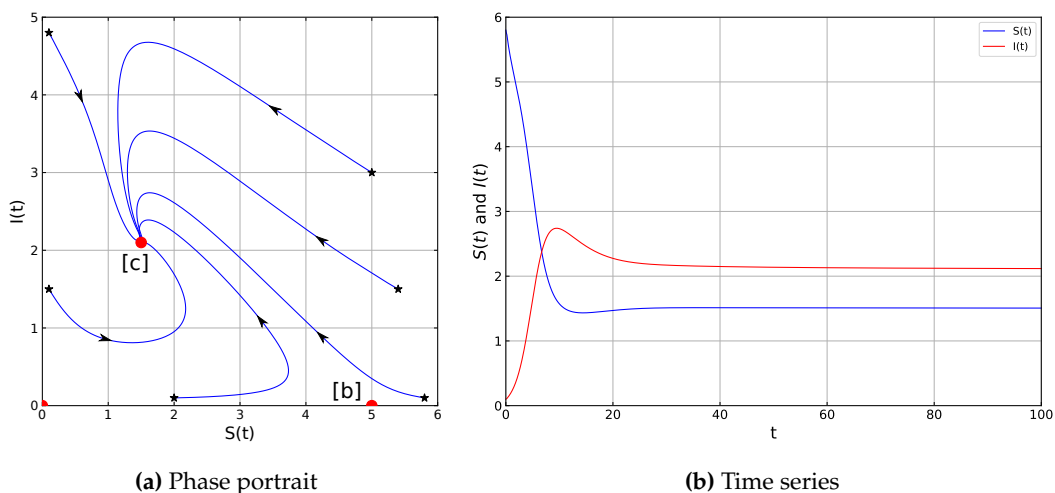**(a)** Phase portrait   **(b)** Time series

Figure 3. Numerical simulation of model (3) with parameter values: $r = 0.5, K = 5, \beta = 0.2 \ \Omega = 0.2$, and $\delta = 0.1$

To show the dynamical behavior of model (3) when $R_0 < 1$ and $R_0 > 1$, we set $\beta = 0.05$ (or $R_0 = 0.83333$) and $\beta = 0.2$ (or $R_0 = 3.33333$), respectively. When $R_0 = 0.83333$, we have an equilibrium point which given by label [a]=(5,0), see Figure 1. The equilibrium point [a] is asymptotically stable both locally and globally as proven in Theorem 4, and shown in Figure 2. For $R_0 = 3.33333$, we have two equilibrium points labeled by [b] and [c], see Figure 1. The equilibrium point [b] is unstable point while the equilibrium point [c] is globally asymptotically stable. The phaseportrait and the time series are shown in Figure 3. From those numerical simulations, we conclude that when $R_0 < 1$, the disease won't spread, and when $R_0 > 1$, the disease will spread and convergent to a constant value. The disease is still exists for $t \to \infty$ but the density of infected population is bounded.

6. Conclusion

The dynamics of a fractional-order logistic growth model with infectious disease has been investigated. We show analytically that the model has atmost three equilibrium points i.e: the population extinction point which is always exists and a saddle point, the disease-free point which is always exists, asymptotically stable when $R_0 < 1$ and a saddle point when $R_0 > 1$, and the endemic point which is exists and asymptotically stable when $R_0 > 1$. We succesfully proof that the global stability has the similar properties as the local one for each non-trivial equilibrium point. We also show there exists a forward bifurcation driven by β or R_0. From biological point of view, if $R_0 < 1$ the disease will extinct, and if $R_0 > 1$, the disease will spreads. Although the disease is spreading, the disease will bounded an confine to a region.

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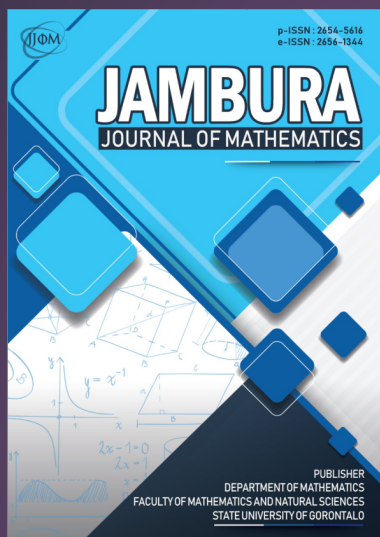
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