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Dynamics of SIS–Epidemic model with Competition Involving Fractional-Order Derivative With Power-Law Kernel

Ismail Djakaria^{a,*}, Hasan S. Panigoro^b, Ebenezer Bonyah^c, Emli Rahmi^b, Wahab Musa^d

^a*Magister Mathematics Education Programme, Post-Graduate, Universitas Negeri Gorontalo, Gorontalo 96128, Indonesia*

^b*Biomathematics Research Group, Department of Mathematics, Faculty of Mathematics and Natural Sciences, Universitas Negeri Gorontalo, Bone Bolango 96119, Indonesia*

^c*Department of Mathematics Education, Akenten Appiah-Menka University of Skills Training and Entrepreneurial Development, Kumasi 00233, Ghana*

^d*Department of Electrical Engineering, Universitas Negeri Gorontalo, Gorontalo 96128, Indonesia*

Abstract

Infectious disease and competition play important roles in the dynamics of a population due to their capability to increase the mortality rate for each organism. In this paper, the dynamical behaviors of a single species population are studied by considering the existence of the infectious disease, intraspecific competition, and interspecific competition. The fractional-order derivative with a power-law kernel is utilized to involve the impact of the memory effect. The population is divided into two compartments namely the susceptible class and the infected class. The existence, uniqueness, non-negativity, and boundedness of the solution are investigated to confirm the biological validity. Three types of feasible equilibrium points are identified namely the origin, the disease-free, and the endemic points. All biological conditions which present the local and global stability are investigated. The global sensitivity analysis is given to investigate the most influential parameter to the basic reproduction number and the density of each class. Some numerical simulations including bifurcation diagrams and time series are also portrayed to explore more the dynamical behaviors.

Keywords: Infectious Disease, Competition, Fractional Derivative, Caputo Operator, Dynamical Behaviors

1. Introduction

The spread of infectious disease still becomes a fundamental issue not only because of the existence of the population but also to maintain the balance of biological systems. Several scientific methods are developed to discover better ways to suppress and control the rate of disease infection [1]. The preferred ways for the last decades for this epidemiological problems are given by mathematical approach using a deterministic model which is considered efficacious to understand the mechanisms of disease transmission and evaluate the appropriate control strategies [2–4]. The fundamental one which has become the basis of epidemiological modeling is given by [5] which develops the continuous-time deterministic model using first-order derivative as the operator. This model is successfully developed in couple of ways such as the continuous-time single species epidemiological modeling with first-order derivative [6–9], the discrete-time single species epidemiological modeling [10–12], the stochastic single-species epidemiological modeling [13, 14], and the continuous-time eco-epidemiological modeling [15–17].

Apart from those operators, several researchers prefer to use the fractional-order derivative to accomplish their problems the biological modeling. See [18–20] and references therein for some examples in epidemiological modeling. The fractional-order derivative is chosen by considering the capability of this operator to describe the current state of the biological object as the impact of all of its previous conditions

*Corresponding author

Email address: iskar@ung.ac.id (Ismail Djakaria)

17 which are known as the memory effect [21, 22]. In the epidemiological model, the transmission of disease
18 may slow down and be forestalled by the susceptible population as the impact of the memory [23]. Some
19 fractional-order derivative has been developed and successfully applied in epidemiological modeling such
20 as the Riemann-Liouville, Caputo, Caputo-Fabrizio, and Atangana-Baleanu [24–27]. From all of the given
21 operators, the Caputo fractional-order derivative has the complete tools for dynamical analysis such as the
22 existence and uniqueness, non-negativity and boundedness, local dynamics, global dynamics, and some bi-
23 furcation analysis. Consequently, the Caputo operator will be used in this paper where defined later in the
24 next section.

25 In this work, we develop the epidemiological model based on the SIR model given by [5]. For single-species
26 conditions, this model is only popular for the infectious diseases that appeared in the human population.
27 In facts, infectious diseases also threaten the existence of the animal population which disturbs the balance
28 of the ecosystem. For examples, the infectious diseases in endemic species such as Orangutans [28], Tarsius
29 [29], Sumatran Tiger [30], and Komodo dragon [31]. Moreover, the natural behaviors of animals that
30 endanger the existence of their populations are the intraspecific competition among them to preserve their
31 food sources [32–34]. For these reasons, developing and investigating the dynamics of the epidemiological
32 model by considering the impact of intraspecific competition and the memory effect are critical issues that
33 become the novelty of our research.

34 The whole of this paper is organized in the following procedure: In Section 2, the mathematical mod-
35 eling consists of model formulation, existence, uniqueness, non-negativity, and boundedness are given. The
36 analytical results including the existence of equilibrium points and their local and global dynamics are com-
37 pletely investigated in Section 3. To show the most influential parameter of the model, the global sensitivity
38 analysis is provided by Section 4. Some numerical simulations as well as bifurcation diagrams and time-series
39 are presented in Section 5 to explore more about the dynamical behaviors of the model. This work ends by
40 giving a conclusion in Section 6.

41 2. Mathematical Modeling

42 This section studies about mathematical modeling consisting of the model formulation, existence, unique-
43 ness, non-negativity, and boundedness of solution. The mathematical model is constructed by a deterministic
44 approach using a differential equation. We first give some assumptions to restrain the model so it does not
45 get too complicated. We next interpret the giving assumptions to the mathematical formula using the first-
46 order derivative as the operator. A diagram is presented to show the impact of each assumption on the
47 flow of population density for each compartment. To involve the impact of the memory effect, the Caputo
48 fractional-order derivative is applied to the model. For the mathematical model's validity, we show that the
49 solution of the model always exists, unique, non-negative, and bounded.

50 2.1. Model Formulation

51 In this work, the model is constructed from a single population growth model. We first assume there
52 exists a population in a habitat that grows proportionally to its density and bounded due to the intraspecific
53 competition. Let $N(t)$ be the population at time t , r is the birth rate, μ is the natural death rate, and ω
54 is the death rate as a result of competition. Thus, we have a first-order differential equation as follows.

$$55 \frac{dN}{dt} = (r - \mu)N - \omega N^2. \quad (1)$$

56 Next, we assume that the population is exposed by infectious disease. The population N is divided into two
57 compartments namely the susceptible class (S) and infected class (I) where $N = S + I$. The susceptible class
58 is infected by disease bilinearly with infection rate β . The competition is divided into two cases namely the
59 intraspecific competition for each susceptible and infected class, and the interspecific competition between
susceptible and infected classes. As result, the following model is received.

$$60 \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - \mu I, \end{aligned} \quad (2)$$

60 where ω_i , $i = 1, 2$ respectively denote the death rate of the susceptible population as the results of intraspe-
 61 cific and interspecific competitions between susceptible and susceptible classes, and susceptible and infected
 62 classes. The parameters ω_i , $i = 3, 4$ denote the death rate of the infected population as the result of com-
 63 petition between infected and infected classes, and susceptible and infected classes. In our works, we also
 64 assume that each organism has the capability to survive the disease. Thus, we define η as the recovery rate.
 65 Since each organism that survives from the disease has a chance to be re-infected, this type of population
 66 will be again susceptible. Finally, we have a mathematical model as follows.

$$\begin{aligned}\frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I.\end{aligned}\tag{3}$$

67 All of the given assumptions and their mathematical modeling are described in Figure 1.

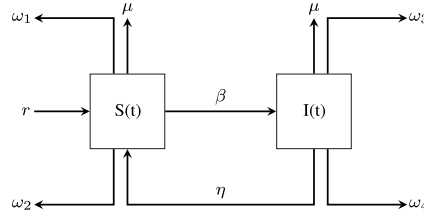


Figure 1: Compartment diagram of model (3)

68 Now, the Caputo fractional-order derivative will be applied in order to conduct the impact of the memory
 69 effect on the population growth rate. The similar procedure is adopted from [35]. The first-order derivatives
 70 on the left-hand side of model (3) are replaced by the Caputo fractional-order derivative defined as follows.

71 **Definition 1.** [36] Suppose $0 < \alpha \leq 1$. The Caputo fractional derivative of order $-\alpha$ is defined by

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

72 where $t \geq 0$, $f \in C^n([0, +\infty), \mathbb{R})$, and Γ is the Gamma function.

73 Applying Definition 1 to eq. (3), the following model is obtained.

$$\begin{aligned}{}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I.\end{aligned}\tag{5}$$

74 Since the given process above makes the dimension of time at the left-hand side become t^α , some parameters
 75 need to be rescaled so that there are no differences between the time's dimensions at the left-hand side with
 76 the right-hand side of model (5). By applying time rescale to some parameters, we have the model as follows.

$$\begin{aligned}{}^C \mathcal{D}_t^\alpha S &= (r^\alpha - \mu^\alpha)S - \omega_1^\alpha S^2 - (\omega_2^\alpha + \beta^\alpha)SI + \eta^\alpha I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta^\alpha - \omega_4^\alpha)SI - \omega_3^\alpha I^2 - (\eta^\alpha + \mu^\alpha)I.\end{aligned}\tag{6}$$

77 Let $r^\alpha = \hat{r}$, $\mu^\alpha = \hat{\mu}$, $\omega_1^\alpha = \hat{\omega}_1$, $\omega_2^\alpha = \hat{\omega}_2$, $\omega_3^\alpha = \hat{\omega}_3$, $\omega_4^\alpha = \hat{\omega}_4$, $\beta^\alpha = \hat{\beta}$, and $\eta^\alpha = \hat{\eta}$. Thus, we acquire

$$\begin{aligned}{}^C \mathcal{D}_t^\alpha S &= (\hat{r} - \hat{\mu})S - \hat{\omega}_1 S^2 - (\hat{\omega}_2 + \hat{\beta})SI + \hat{\eta}I, \\ {}^C \mathcal{D}_t^\alpha I &= (\hat{\beta} - \hat{\omega}_4)SI - \hat{\omega}_3 I^2 - (\hat{\eta} + \hat{\mu})I.\end{aligned}\tag{7}$$

78 For simplicity, by dropping $\hat{\cdot}$ for each parameter, we obtain the final model as follows.

$$\begin{aligned}{}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I = F_1(N(t)), \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I = F_2(N(t)).\end{aligned}\tag{8}$$

79 Equation (8) is the final proposed model in this paper. Although model (8) seems classic and simple,
80 this model will be powerful to solve and investigate the existence of a closed population in a certain area
81 without any outside intervention. Our literature review also shows that the model (8) has heretofore never
82 been studied. Now, the basic properties of model (8) such as the existence uniqueness, non-negativity, and
83 boundedness are investigated to confirm its biological validity.

84 2.2. Existence and Uniqueness

85 In this subsection, we will show that the model (8) has a unique solution. A similar manner given by
86 [37] is used. Thus, the following theorem is presented to show the existence and uniqueness of the solution
87 of model (8).

88 **Theorem 1.** *The model (8) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ has a unique solution.*

89 **Proof.** Consider model (8) with positive initial condition with $F : [0, \infty) \rightarrow \mathbb{R}^2$ where $F(N) = (F_1(N), F_2(N))$,
90 $N \equiv N(t)$ and $\theta \equiv \{(S, I) \in \mathbb{R}_+^2 : \max\{|S|, |I|\} \leq M\}$ for sufficiently large M . Then, for any $N = (S, I)$
91 and $\bar{N} = (\bar{S}, \bar{I})$, $N, \bar{N} \in \theta$, we have

$$\begin{aligned}
\|F(N) - F(\bar{N})\| &= |F_1(N) - F_1(\bar{N})| + |F_2(N) - F_2(\bar{N})| \\
&= |[(r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I] - [(r - \mu)\bar{S} - \omega_1 \bar{S}^2 - (\omega_2 + \beta)\bar{S}\bar{I} + \eta \bar{I}]| + \\
&\quad |[(\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I] - [(\beta - \omega_4)\bar{S}\bar{I} - \omega_3 \bar{I}^2 - (\eta + \mu)\bar{I}]| \\
&\leq (r + \mu) |S - \bar{S}| + \omega_1 |S^2 - \bar{S}^2| + (\omega_2 + \beta) |SI - \bar{S}\bar{I}| + \eta |I - \bar{I}| + (\beta + \omega_4) |SI - \bar{S}\bar{I}| \\
&\quad + \omega_3 |I^2 - \bar{I}^2| + (\eta + \mu) |I - \bar{I}| \\
&= (r + \mu) |S - \bar{S}| + \omega_1 |(S + \bar{S})(S - \bar{S})| + (\omega_2 + \omega_4 + 2\beta) |I(S - \bar{S}) + \bar{S}(I - \bar{I})| \\
&\quad + (2\eta + \mu) |I - \bar{I}| + \omega_3 |(I + \bar{I})(I - \bar{I})| \\
&\leq (r + \mu) |S - \bar{S}| + 2\omega_1 M |S - \bar{S}| + (\omega_2 + \omega_4 + 2\beta) M |S - \bar{S}| \\
&\quad + (\omega_2 + \omega_4 + 2\beta) M |I - \bar{I}| + (2\eta + \mu) |I - \bar{I}| + 2\omega_3 M |I - \bar{I}| \\
&= [(r + \mu) + 2\omega_1 M + (\omega_2 + \omega_4 + 2\beta) M] |S - \bar{S}| + [(\omega_2 + \omega_4 + 2\beta) M + (2\eta + \mu) + 2\omega_3 M] |I - \bar{I}| \\
&\leq L \|N - \bar{N}\|,
\end{aligned}$$

92 where $L = (\omega_2 + \omega_4 + 2\beta) M + \mu + \max\{r + 2\omega_1 M, 2(\eta + \omega_3 M)\}$. Therefore, $F(N)$ satisfies the Lipschitz
93 condition. Obeying Lemma 5 in [38], we conclude that model (8) with positive initial condition has a unique
94 solution. \square

95 2.3. Non-negativity and Boundedness

96 The non-negativity and boundedness properties of the solutions of the model (8) are given in the following
97 theorem.

98 **Theorem 2.** *All solution of the model (8), which start in $\mathbb{R}_+^2 := \{(S, I) \mid S \geq 0, I \geq 0, (S, I) \in \mathbb{R}^2\}$ are
99 uniformly bounded and non-negative.*

100 **Proof.** To prove the boundedness of the solutions of the model (8), the same approach of [38] is adopted.
101 Let consider the function $N = S + I$. Then,

$$\begin{aligned}
{}^C \mathcal{D}_t^\alpha N &= {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I + (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I.
\end{aligned}$$

102 Hence, for each $\mu > 0$,

$${}^C \mathcal{D}_t^\alpha N + \mu N = (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I + \mu S + \mu I$$

$$\begin{aligned}
&= rS - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&= -\omega_1 \left(S - \frac{r}{2\omega_1} \right)^2 + \frac{r^2}{4\omega_1} - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&\leq \frac{r^2}{4\omega_1}
\end{aligned}$$

103 By using the comparison theorem in [39], we obtain $N(t) \leq N(0)E_\alpha(-\mu t^\alpha) + \frac{r^2}{4\omega_1} t^\alpha E_{\alpha, \alpha+1}(-\mu t^\alpha)$, where
104 E_α and $E_{\alpha, \alpha+1}$ is the Mittag-Leffler function with one and two parameters. According to Lemma 5 and
105 Corollary 6 in [39], we have $N(t) \leq \frac{r^2}{4\mu\omega_1}$, as $t \rightarrow \infty$. Therefore, all solutions of model (8) starting in
106 \mathbb{R}_+^2 are uniformly bounded in the region Φ , where $\Phi = \left\{ (S, I) \in \mathbb{R}_+^2 : S + I \leq \frac{r^2}{4\mu\omega_1} + \epsilon, \epsilon > 0 \right\}$. Next, we
107 prove that all solutions of model (8) are non-negative. By model (8), we have ${}^C\mathcal{D}_t^\alpha S|_{S=0} = \eta I \geq 0$ and
108 ${}^C\mathcal{D}_t^\alpha I|_{I=0} = 0 \geq 0$. Based on Lemmas 5 and 6 in [40], we conclude that the solutions of model (8) are
109 non-negative. \square

110 3. Analytical Results

111 In this section, the dynamics of model (8) are shown analytically including the existence of equilibrium
112 points, and their local and global stability.

113 3.1. Existence of Equilibrium Points

114 To find the equilibrium points of model (8), we must have

$$[(r - \mu) - \omega_1 S - (\omega_2 + \beta)I]S + \eta I = 0, \quad (9)$$

$$[(\beta - \omega_4)S - \omega_3 I - (\eta + \mu)]I = 0. \quad (10)$$

115 If $I = 0$ is substituted to (9), we obtain

$$[(r - \mu) - \omega_1 S]S = 0. \quad (11)$$

116 From eq. (11), we get $S = 0$ and $S = \frac{r - \mu}{\omega_1}$. Thus, we have two equilibrium points here namely $\mathcal{E}_0 = (0, 0)$,
117 and $\mathcal{E}_A = \left(\frac{r - \mu}{\omega_1}, 0 \right)$. The equilibrium point \mathcal{E}_0 is called the origin point which represents the extinction of
118 both susceptible and infected populations. Since $\mathcal{E}_0 \in \mathbb{R}_+^2$, this equilibrium point always exists. Furthermore,
119 the equilibrium point \mathcal{E}_A is called the disease-free equilibrium point (DFEP) which describes the condition
120 where the infectious disease does not exist anymore in the population. According to the biological condition,
121 it is natural that the birth rate r is greater than its death rate μ . By assuming $r > \mu$, the origin point
122 $\mathcal{E}_A \in \mathbb{R}_+^2$ also always exists. By simple calculation, we also obtain the basic reproduction number \mathcal{R}_0 given
123 by

$$\mathcal{R}_0 = \frac{(r - \mu)\beta}{(r - \mu)\omega_4 + (\eta + \mu)\omega_1}. \quad (12)$$

124 The basic reproduction number is utilized to show the dynamical behavior of each equilibrium point and
125 to describe whether the infectious disease becomes endemic or not. Since $r > \mu$, the value of \mathcal{R}_0 is always
126 positive. Now, let's concern the eq. (9) and (10). By solving eq. (10), we attain

$$S = \frac{\omega_3 I + (\eta + \mu)}{\beta - \omega_4}. \quad (13)$$

127 If we substitute eq. (13) to (9), the following polynomial equation holds.

$$k_1 I^2 + k_2 I + k_3 = 0, \quad (14)$$

128 where

$$\begin{aligned}
k_1 &= ((\beta - \omega_4)(\beta + \omega_2) + \omega_1\omega_3)\omega_3, \\
k_2 &= (\beta - \omega_4)((\beta + \omega_2)\mu + (\omega_2 + \omega_4)\eta - (r - \mu)\omega_3) + 2(\eta + \mu)\omega_1\omega_3, \\
k_3 &= \frac{(1 - \mathcal{R}_0)(r - \mu)(\eta + \mu)\beta}{\mathcal{R}_0}.
\end{aligned}$$

129 Therefore, we acquire the endemic point (EEP)

$$\mathcal{E}_I = \left(\frac{\omega_3\bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}, \bar{\gamma} \right), \quad (15)$$

130 where $\bar{\gamma}$ is the positive root of polynomial equation (14). From (15), we find that $\beta > \omega_4$ must be fulfilled so
131 that $\mathcal{E}_I \in \mathbb{R}_+^2$. Moreover, EEP exists if $\bar{\gamma} > 0$. From eq. (14), we have k_1 is always positive. Thus, the value
132 of the $\bar{\gamma}$ depends on k_2 and k_3 . Furthermore, eq. (14) has real number roots if $k_2^2 \geq 4k_1k_3$. By applying
133 simple algebra, if $k_3 > 0$ and $k_2 < 0$ then we have two positive roots of eq. (14), if $k_3 > 0$ and $k_2 > 0$ then
134 we do not have any positive roots of eq. (14), and if $k_3 < 0$ then we have a positive root of eq. (14). Finally,
135 we have the following theorem.

136 **Theorem 3.** *Let $\beta > \omega_4$. The existence of EEP \mathcal{E}_I is shown by the following statement.*

- 137 (i) *If $k_2^2 < 4k_1k_3$ then \mathcal{E}_I does not exist.*
138 (ii) *If $k_2^2 = 4k_1k_3$ and*
139 *(ii.i) if $k_2 > 0$ then \mathcal{E}_I does not exist.*
140 *(ii.ii) if $k_2 < 0$ then \mathcal{E}_I exists and unique.*
141 (iii) *If $k_2^2 > 4k_1k_3$ and*
142 *(iii.i) if $k_3 > 0$ and $k_2 < 0$ then we have a pair of \mathcal{E}_I .*
143 *(iii.ii) if $k_3 > 0$ and $k_2 > 0$ then \mathcal{E}_I does not exist.*
144 *(iii.iii) if $k_3 < 0$ then \mathcal{E}_I exists and unique.*

145 Denote that $k_2^2 > 4k_1k_3$ is always satisfied and $k_3 < 0$ for $\mathcal{R}_0 > 1$, then the following lemma holds.

146 **Lemma 4.** *EEP \mathcal{E}_I exists and unique if $\mathcal{R}_0 > 1$.*

147 3.2. Local Dynamics

148 The local dynamics of model (8) are obtained by applying the Matignon condition which is defined as
149 follows.

150 **Theorem 5.** *[Matignon condition [36]] An equilibrium point \bar{x}^* is locally asymptotically stable (LAS) if
151 all eigenvalues λ_j of the Jacobian matrix $J = \frac{\partial \bar{f}}{\partial \bar{x}}$ at \bar{x}^* satisfy $|\arg(\lambda_j)| > \frac{\alpha\pi}{2}$. If there exists at least one
152 eigenvalue satisfy $|\arg(\lambda_k)| > \frac{\alpha\pi}{2}$ while $|\arg(\lambda_l)| < \frac{\alpha\pi}{2}$, $k \neq l$, then \bar{x}^* is a saddle-point.*

153 Therefore, to study the local dynamics of model (8), we first compute its Jacobian matrix at the point
154 (S, I) which gives

$$\mathcal{J}(S, I) = \begin{bmatrix} (r - \mu) - 2\omega_1 S - (\omega_2 + \beta)I & -(\omega_2 + \beta)S + \eta \\ (\beta - \omega_4)I & (\beta - \omega_4)S - 2\omega_3 I - (\eta + \mu) \end{bmatrix}. \quad (16)$$

155 Obeying Theorem 5 and using Jacobian matrix (16), we discuss the local stability for each equilibrium point
156 in the next subsection.

157 *3.3. Dynamical behavior around \mathcal{E}_0*

158 LAS condition of \mathcal{E}_0 is obtained by identifying the eigenvalues of the Jacobian matrix (16) at the point
159 $(S, I) = (0, 0)$. We receive

$$\mathcal{J}(S, I)|_{\mathcal{E}_0} = \begin{bmatrix} r - \mu & \eta \\ 0 & -(\eta + \mu) \end{bmatrix}.$$

160 Therefore, we have $\lambda_1 = r - \mu$ and $\lambda_2 = -(\eta + \mu)$. Since $r > \mu$ and $\lambda_2 < 0$, we have $|\arg(\lambda_1)| = 0 < \frac{\alpha\pi}{2}$
161 and $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$. According to Theorem 5, the following theorem holds.

162 **Theorem 6.** *The origin point \mathcal{E}_0 is always a saddle point.*

163 *3.4. Dynamical behavior around \mathcal{E}_A*

For $(x, y) = \left(\frac{r-\mu}{\omega_1}, 0\right)$, the Jacobian matrix (16) becomes

$$\mathcal{J}(S, I)|_{\mathcal{E}_A} = \begin{bmatrix} -(r - \mu) & \eta - \frac{(\omega_2 + \beta)(r - \mu)}{\omega_1 \mathcal{R}_0} \\ 0 & \frac{(\mathcal{R}_0 - 1)(r - \mu)\beta}{\omega_1 \mathcal{R}_0} \end{bmatrix},$$

164 which gives a pair of eigenvalues $\lambda_1 = -(r - \mu)$ and $\lambda_2 = \frac{(\mathcal{R}_0 - 1)(r - \mu)\beta}{\omega_1 \mathcal{R}_0}$. Denote $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$ as the
165 impact of $\lambda_1 < 0$. Hence, the sign of λ_2 takes the role in describing local dynamics around \mathcal{E}_A . To obtain
166 $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$, we need $\lambda_2 < 0$ which is fulfilled if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ then $|\arg(\lambda_2)| = 0 < \frac{\alpha\pi}{2}$.
167 Following the Matignon condition given in Theorem 5, the following theorem is successfully attained.

168 **Theorem 7.** *If $\mathcal{R}_0 < 1$ then \mathcal{E}_A is LAS and a saddle point if $\mathcal{R}_0 > 1$.*

169 *3.5. Dynamical behavior around \mathcal{E}_I*

170 To identify the local stability of \mathcal{E}_I , we first compute the Jacobian matrix (16) evaluated at \mathcal{E}_I . We
171 generate

$$\mathcal{J}(S, I)|_{\mathcal{E}_I} = \begin{bmatrix} - \left[\frac{(\omega_3 \bar{\gamma} + \eta + \mu)\omega_1}{\beta - \omega_4} + \frac{(\beta - \omega_4)\eta \bar{\gamma}}{\omega_3 \bar{\gamma} + \eta + \mu} \right] & - \frac{(\omega_2 + \beta)(\omega_3 \bar{\gamma} + \eta + \mu)}{\beta - \omega_4} + \eta \\ (\beta - \omega_4)\bar{\gamma} & -\omega_3 \bar{\gamma} \end{bmatrix}. \quad (17)$$

172 The eigenvalues of (17) are given by $\lambda_1 = \frac{1}{2} \left(\xi_1 + \sqrt{\xi_1^2 - 4\xi_2} \right)$ and $\lambda_2 = \frac{1}{2} \left(\xi_1 - \sqrt{\xi_1^2 - 4\xi_2} \right)$ where

$$\begin{aligned} \xi_1 &= - \left[\frac{(\omega_3 \bar{\gamma} + \eta + \mu)\omega_1}{\beta - \omega_4} + \frac{(\beta - \omega_4)\eta \bar{\gamma}}{\omega_3 \bar{\gamma} + \eta + \mu} + \omega_3 \bar{\gamma} \right], \\ \xi_2 &= \left[\left(\frac{\omega_1 \omega_3}{\beta - \omega_4} + \omega_2 + \beta \right) (\omega_3 \bar{\gamma} + \eta + \mu) + \left(\frac{\omega_3 \bar{\gamma}}{\omega_3 \bar{\gamma} + \eta + \mu} + 1 \right) (\beta - \omega_4)\eta \right] \bar{\gamma}. \end{aligned}$$

173 It is easy to proof that $\xi_1 < 0$ and $\xi_2 > 0$ since $\beta > \omega_4$ becomes the existence condition. As the impact,
174 $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$, $i = 1, 2$ and hence the LAS always hold for EEP. Thus, the following theorem holds.

175 **Theorem 8.** *EEP \mathcal{E}_I is always LAS.*

176 *3.6. Global Dynamics*

177 In this subsection, the global dynamics of model (8) are studied. The biological conditions of equilibrium
178 points are investigated so that those points are globally asymptotically stable (GAS). Since the origin is
179 always a saddle point, we focus on studying GAS conditions for DFEP and EEP. The next two theorems
180 are given for the global dynamics.

181 **Theorem 9.** *DFEP \mathcal{E}_A is GAS if $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$.*

182 **Proof.** We define a positive Lyapunov function as follows.

$$\mathcal{V}_A(S, I) = \left(S - \frac{r - \mu}{\omega_1} - \frac{r - \mu}{\omega_1} \ln \frac{\omega_1 S}{r - \mu} \right) + I. \quad (18)$$

183 If we calculate the Caputo fractional derivative of $\mathcal{V}_A(S, I)$ along the solution of model (8) and use Lemma
184 3.1 in [41], we get

$$\begin{aligned} {}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) &= \left(\frac{S - \frac{r - \mu}{\omega_1}}{S} \right) {}^C\mathcal{D}_t^\alpha S + {}^C\mathcal{D}_t^\alpha I \\ &= -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 + \frac{(r - \mu)(\omega_2 + \beta)I}{\omega_1} - \frac{(r - \mu)\eta I}{\omega_1 S} - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I \\ &\leq -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 - \left(\mu - \frac{(\omega_2 + \beta)r}{\omega_1} \right) I \end{aligned}$$

185 Since $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$, we have ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$, and ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$ only when
186 $(S, I) = \left(\frac{r - \mu}{\omega_1}, 0 \right)$. This means that the singleton $\{\mathcal{E}_A\}$ is the only invariant set where ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$.
187 By Lemma 4.6 in [42], we can conclude that every solution of model (8) tends to DFEP \mathcal{E}_A .
188 □

189 **Theorem 10.** *EEP \mathcal{E}_I is GAS if $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$.*

190 **Proof.** We first define $\vartheta = \frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}$ and hence $\mathcal{E}_I = (\vartheta, \bar{\gamma})$. Now, a positive Lyapunov function is presented
191 as follows.

$$\mathcal{V}_I(S, I) = \left(S - \vartheta - \vartheta \ln \frac{S}{\vartheta} \right) + \left(I - \bar{\gamma} - \bar{\gamma} \ln \frac{I}{\bar{\gamma}} \right) \quad (19)$$

192 Following Lemma 3.1 in [41], we reach

$$\begin{aligned} {}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) &= \left(\frac{S - \vartheta}{S} \right) {}^C\mathcal{D}_t^\alpha S + \left(\frac{I - \bar{\gamma}}{I} \right) {}^C\mathcal{D}_t^\alpha I \\ &= (S - S^*) \left((r - \mu) - \omega_1 S - (\omega_2 + \beta)I + \frac{\eta I}{S} \right) + (I - \bar{\gamma}) \left((\beta - \omega_4)S - \omega_3 I - (\eta + \mu) \right) \\ &= -\omega_1 (S - \vartheta)^2 - \omega_3 (I - \bar{\gamma})^2 - (\omega_2 + \omega_4) (S - S^*) (I - \bar{\gamma}) \\ &\leq -\left(\omega_1 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} \right) \right) (S - \vartheta)^2 - \left(\omega_3 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} \right) \right) (I - \bar{\gamma})^2 \end{aligned}$$

193 Denote that ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$ as a result of $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$. We also have
194 that ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$ only when $(S, I) = (\vartheta, \bar{\gamma})$. Therefore, the singleton $\{\mathcal{E}_I\}$ is the only invariant set
195 where ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$. Obeying Lemma 4.6 in [42], every solution of model (8) tends to EEP \mathcal{E}_I . □

196 4. Global Sensitivity Analysis

197 In this section, the global sensitivity analysis is studied to investigate the most influential parameters
198 of model (8). Global sensitivity analysis is calculated using Partial Rank Coefficient Correlation (PRCC)
199 [43], where the random data processed in PRCC is generated using Saltelli sampling [44]. Two biological
200 components become the objective function for the PRCC namely the basic reproduction number (\mathcal{R}_0) and
201 the population density of infected class ($I(t)$). We first investigate the most influential parameter to the
202 basic reproduction number (\mathcal{R}_0). From eq. (12), we acquire that only r , μ , ω_1 , ω_4 , and η have the influence

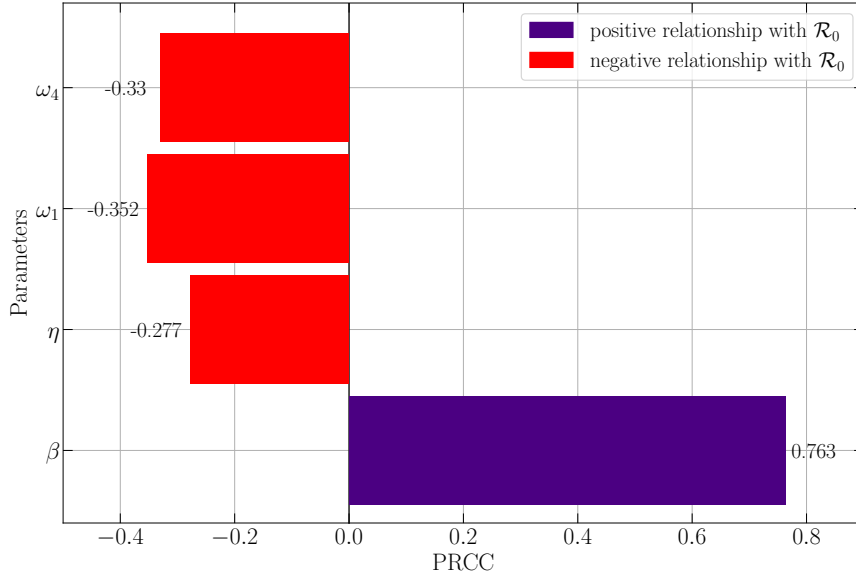


Figure 2: PRCC results for the parameters of \mathcal{R}_0

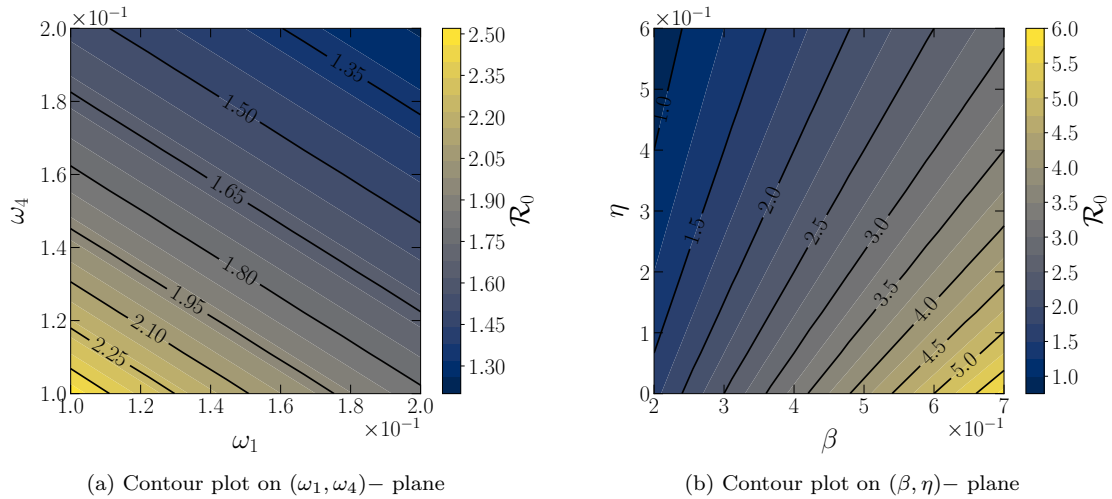


Figure 3: Contour plots for the parameters respect to \mathcal{R}_0

203 on the value of \mathcal{R}_0 . The birth rate and the natural death rate also can be fixed since some cases in the
 204 epidemiological model has the values of these parameters. Thus, only β , η , ω_1 , and ω_4 will be computed for
 205 PRCC. The Figure 2 is given for the results. We have $\beta = 0.763$, $\omega_1 = -0.352$, $\omega_4 = -0.33$, and $\eta = -0.277$
 206 as the coefficient correlation such that the infection rate (β) becomes the most influential parameter to \mathcal{R}_0
 207 and followed by ω_1 , ω_4 , and η , respectively. It shows that the infection rate (β) as the most influential
 208 parameter has a positive relationship with the basic reproduction number (\mathcal{R}_0) which means that \mathcal{R}_0 will
 209 significantly increases when β increases. The rest ω_1 , ω_4 , and η have a negative relationship with \mathcal{R}_0 which
 210 means that by reducing the value of those parameters, the basic reproduction number (\mathcal{R}_0) will increases.
 211 To show the impact of these parameters on \mathcal{R}_0 , the contour plots are also portrayed in Figure 3.

212 Next, we identify the most influential parameter to the population density of infected class ($I(t)$). Quite
 213 similar to previous work, the value of r and μ are fixed but the rest of the parameters are involved to

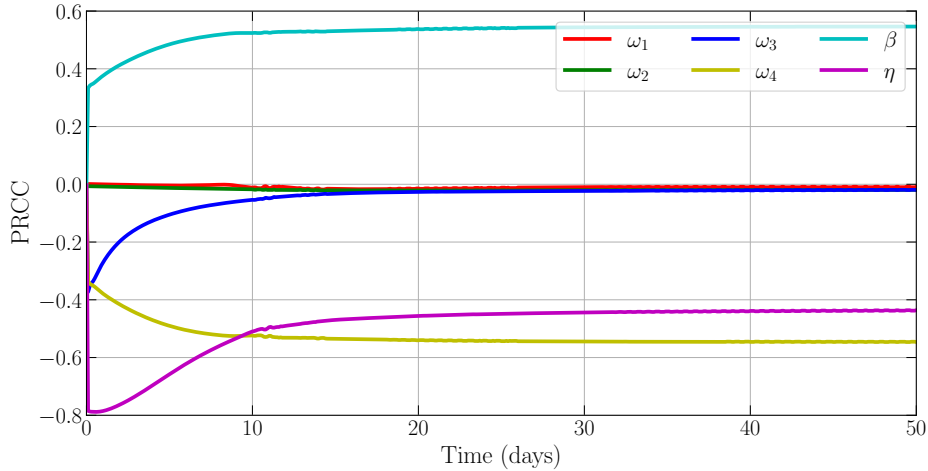


Figure 4: PRCC results for the parameters of $I(t)$

Table 1: PRCC results in respect to the population density of infected class

Parameter	Description	PRCC	Rank	Relationship with $I(t)$
ω_1	The death rate of susceptible population due to the intraspecific competition	-0.00851	6	Negative relationship
ω_2	The death rate of susceptible population due to the interspecific competition	-0.01938	5	Negative relationship
ω_3	The death rate of infected population due to the intraspecific competition	-0.01990	4	Negative relationship
ω_4	The death rate of infected population due to the interspecific competition	-0.54635	1	Negative relationship
β	The infection rate	0.54631	2	Positive relationship
η	The recovery rate	-0.43606	3	Negative relationship

214 compute PRCC. PRCC values are computed for $0 \leq t \leq 50$ which is considered sufficient enough to see the
215 convergence for each parameter through the PRCC. We portray the PRCC results in Figure 4 while the
216 PRCC values, ranks, and the relationship between each parameter and $I(t)$ are given in Table 1. From those
217 simulations, we conclude that the death rate of infected population due to interspecific competition between
218 susceptible and infected classes (ω_4) become the most influential parameter to the population density ($I(t)$)
219 followed respectively by β , η , ω_3 , ω_2 , and ω_1 . In the next section, the numerical simulations including
220 bifurcation diagram and time-series are presented to show the impact of the infection rate (β), recovery
221 rate (η), intraspecific competition (ω_1 and ω_3), and interspecific competition (ω_2 and ω_4) to the dynamical
222 behaviors of model (8).

223 5. Numerical Simulations

224 In this section, the dynamical behaviors of model (8) including bifurcation diagram and time-series are
225 studied numerically. To obtain the bifurcation diagram and the corresponding time-series of model (8),
226 the predictor-corrector scheme developed by Diethelm et al. is employed [45]. Since the model does not
227 investigate a specific epidemiological case, we use hypothetical parameters for all numerical simulations. we
228 set the parameter values as follows.

$$r = 0.6, \mu = 0.1, \omega_1 = 0.1, \omega_2 = 0.1, \omega_3 = 0.1, \omega_4 = 0.1, \beta = 0.4, \eta = 0.2, \text{ and } \alpha = 0.9 \quad (20)$$

229 We start our work by investigating the impact of infection rate (β) on the dynamics of model (8). The
230 value of β is varied in the interval $0 \leq \beta \leq 1$ and we then compute the numerical solutions. To obtain the

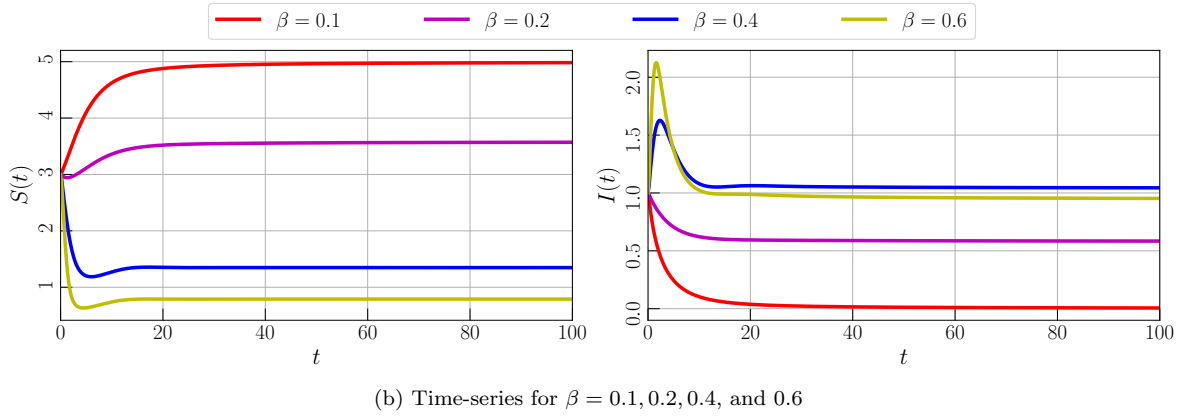
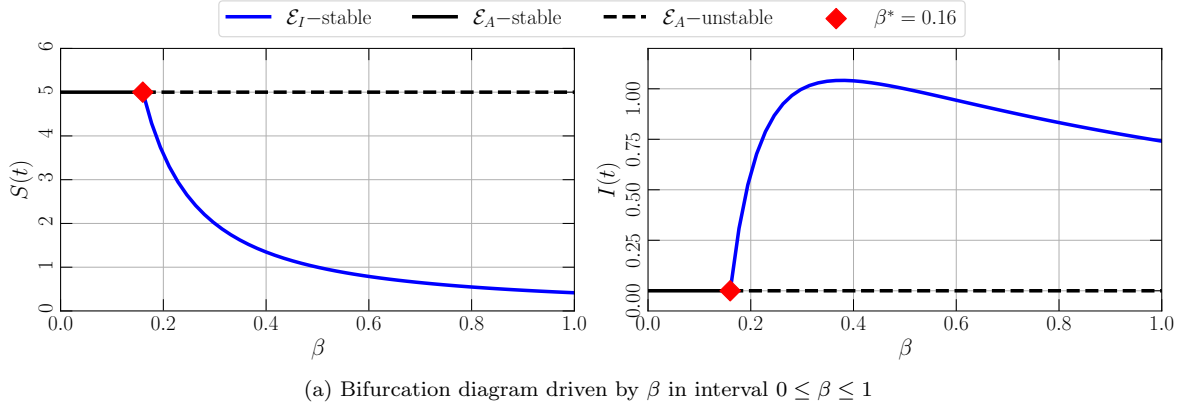
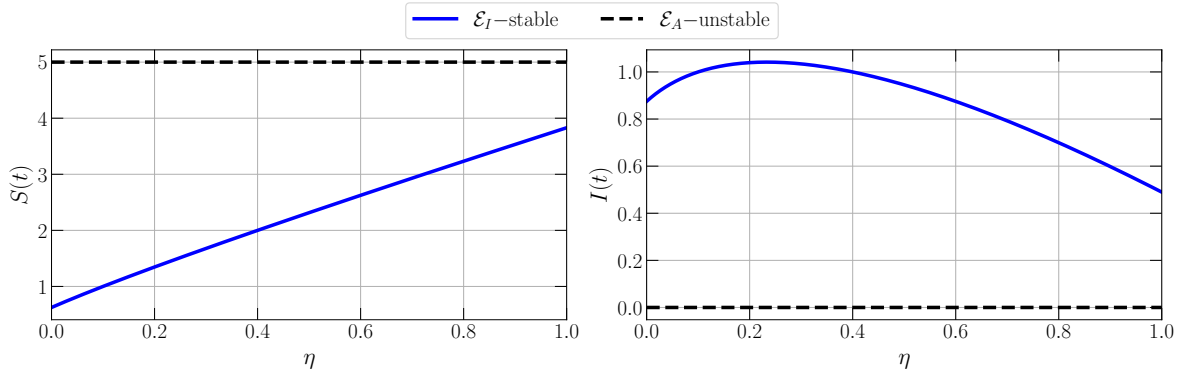


Figure 5: Bifurcation diagram and times-series of model (8) driven by the infection rate (β) with parameter values given by eq. (20)

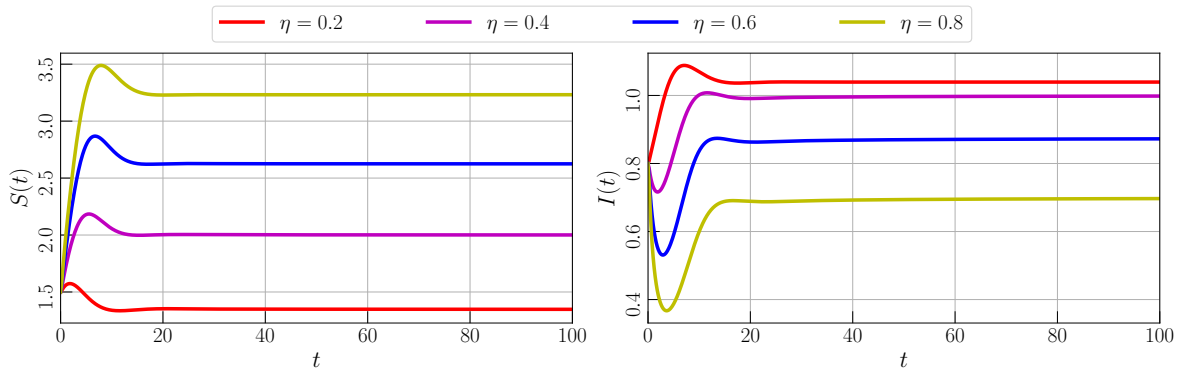
231 bifurcation diagram, we plot the tail of solutions for each β together with the LAS condition of \mathcal{E}_A . As
 232 result, we obtain a bifurcation diagram as in Figure 5a. When $0 \leq \beta < \beta^*$, $\beta^* = 0.16$, the EEP \mathcal{E}_I does not
 233 exist and Theorem 7 is satisfied which means that DFE \mathcal{E}_A is LAS. The solution is convergent to \mathcal{E}_A which
 234 indicates the population free from disease. When β passes through β^* , \mathcal{E}_A losses its stability, and unique
 235 LAS EEP \mathcal{E}_I occurs in the interior. The infectious disease becomes endemic in the population and still
 236 exists for all $t \rightarrow \infty$. From the concatenation of those biological circumstances, we conclude that forward
 237 bifurcation occurs around \mathcal{E}_A where β is the bifurcation parameter and $\beta = \beta^*$ is the bifurcation point. It
 238 is easy to examine that the bifurcation point $\beta = \beta^*$ is equal to $\mathcal{R}_0 = 1$. The dynamical behaviors are
 239 maintained for $\beta^* < \beta \leq 1$. To support these conditions, some time series are given in Figure 5b to show
 240 the convergence of solutions for different values of β .

241 Next, the impact of recover rate (η) is studied. A similar numerical scheme as the previous way is applied.
 242 To depicts the bifurcation diagram, the parameter is fixed as in eq. (20) and the recovery rate (η) is varied
 243 in interval $0 \leq \eta \leq 1$. We have Figure 6a as the result. Denote that the bifurcation does not exist for this
 244 interval. Both DFEP and EEP exist with distinct stability. The DFEP \mathcal{E}_A is a saddle point while the EEP
 245 \mathcal{E}_I is LAS which confirm the validity of Theorems 6 and 7. We also confirm that the EEP \mathcal{E}_I attains GAS
 246 which means that all initial conditions will go right to the EEP and the infectious disease will exist all the
 247 time. Although the disease becomes endemic, the numerical simulation shows that the value of η is directly
 248 proportional to $S(t)$ and inversely proportional to $I(t)$, see Figure 6b. This means the population density
 249 of the infected class can be reduced by increasing the recovery rate (η).

250 For the next simulation, the impact of intraspecific competition is investigated. The death rate parame-



(a) Bifurcation diagram driven by η in interval $0 \leq \eta \leq 1$



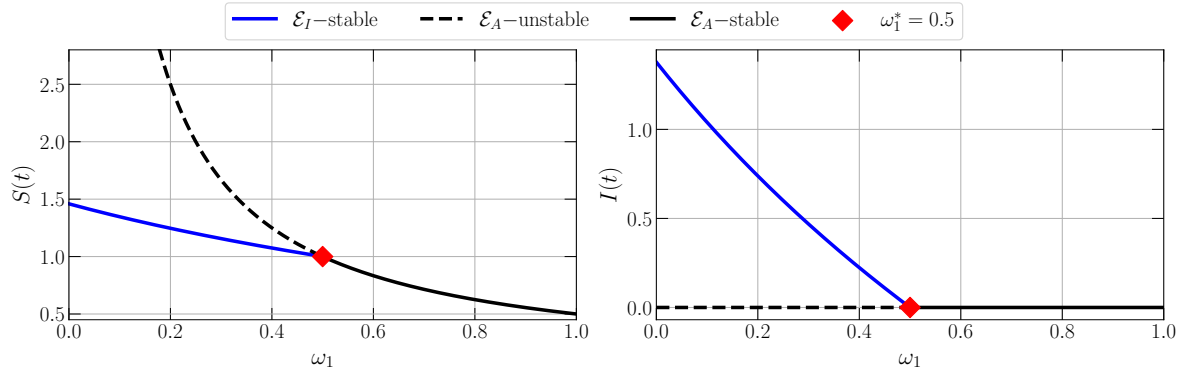
(b) Time-series for $\eta = 0.2, 0.4, 0.6,$ and 0.8

Figure 6: Bifurcation diagram and times-series of model (8) driven by the recovery rate (η) with parameter values given by eq. (20)

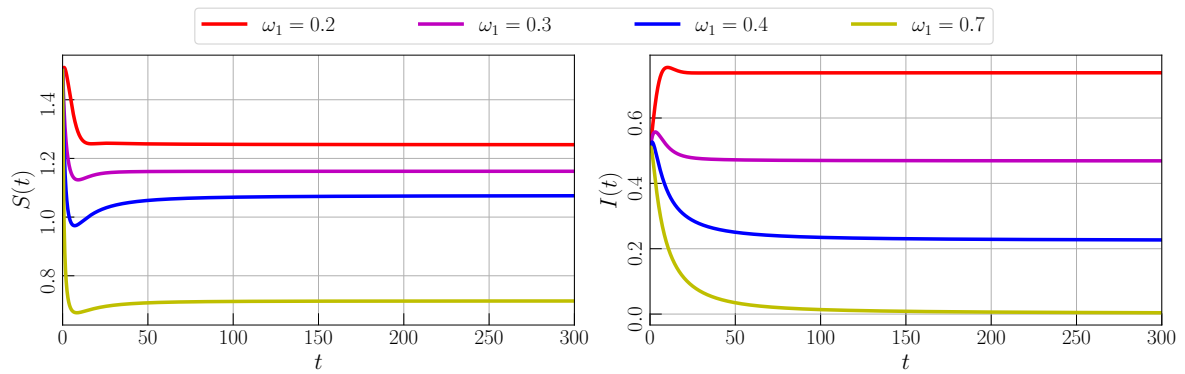
251 ters caused by intraspecific competition on susceptible and infected classes (ω_1 and ω_3) are varied in interval
 252 $[0, 1]$. It is found that forward bifurcation occurs when ω_1 is driven where the bifurcation point is given by
 253 $\omega_1^* = 0.5$, see Figure 7a. The population density of both susceptible and infected classes reduces when the
 254 death rate of $S(t)$ due to intraspecific competition increases as given by Figure 7b. Particularly, Figure 8a
 255 shows that bifurcation does not exist in interval $0 \leq \omega_1 \leq 1$ when ω_1 is varied but the dynamical behaviors
 256 show that $S(t)$ increases and $I(t)$ decrease when ω_1 increase. We confirm this condition by giving time-series
 257 in Figure 8b.

258 Now, we study the impact of interspecific competition on the dynamical behaviors of model (8). Both
 259 susceptible and infected classes have died due to the existence of interspecific competition given by param-
 260 eters ω_2 and ω_4 . By varying ω_2 and ω_4 in interval $[0, 1]$, we obtain Figures 9a and 10a as the bifurcation
 261 diagram. We find forward bifurcation driven by ω_4 which does not exist when varying ω_1 . This means, the
 262 EEP still exists and LAS for $0 \leq \omega_2 \leq 1$. The EEP will disappear via forward bifurcation and the saddle
 263 DFEP becomes LAS when ω_4 crosses $\omega_4^* = 0.34$. This guarantees that the infectious disease may eliminate
 264 the disease in population when the death rate of the infected population due to interspecific competition
 265 increases as shown in Figure 10b. Although the disease does not disappear when ω_2 is driven, we also can
 266 see in Figure 9b that by increasing ω_2 , the population density of the infected class will reduce and the
 267 susceptible class will increase.

268 Finally, the impact of memory effect (α) is investigated. The numerical simulation is given by Figure 11.
 269 For $\alpha = 0.7, 0.8, 0.9, 1$ and similar initial values, all solution converge to single equilibrium point given by
 270 $\mathcal{E}_I \approx (1.3465, 1.0395)$, see Figure 11(a,b). We then plot the local amplification to show the difference of



(a) Bifurcation diagram driven by ω_1 in interval $0 \leq \omega_1 \leq 1$



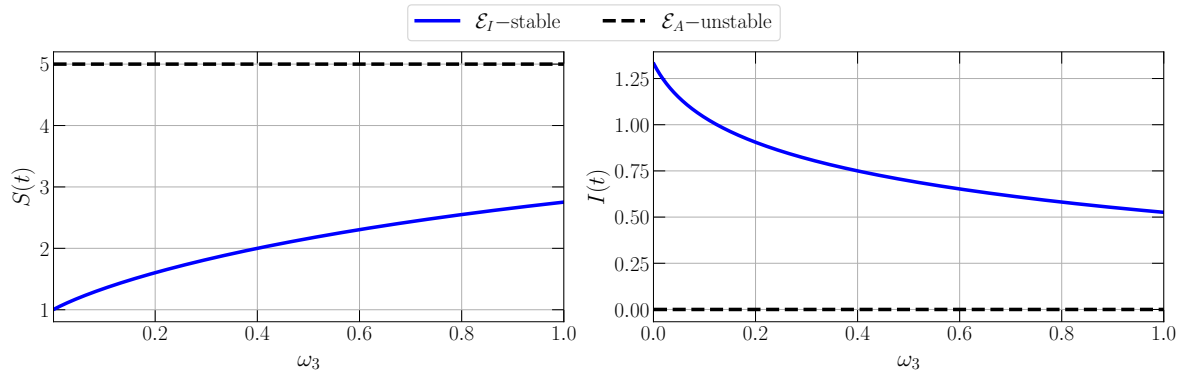
(b) Time-series for $\omega_1 = 0.2, 0.3, 0.4,$ and 0.7

Figure 7: Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to intraspecific competition (ω_1) with parameter values given by eq. (20)

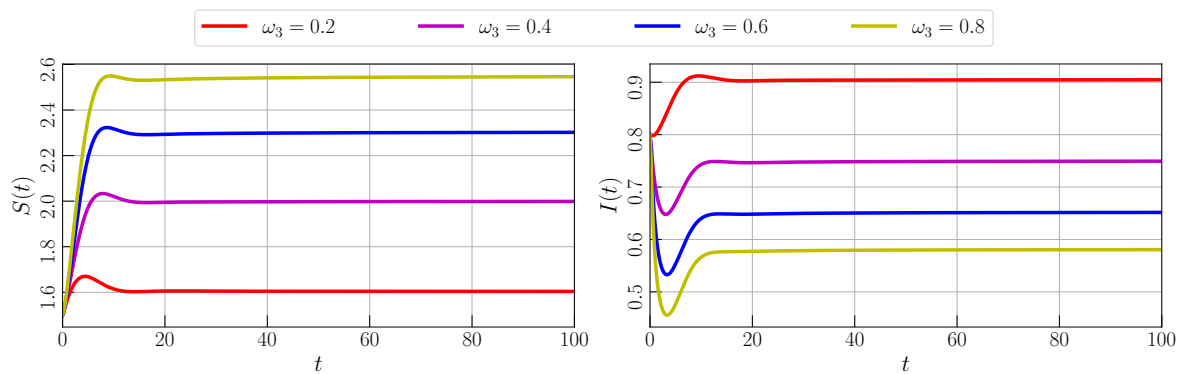
271 solutions when α is varied. We find that the difference lies in the convergence rate where for larger values of
 272 α , the convergence rate increase and vice versa as shown in Figure 11(e,f). In the beginning, Figure 11(c,d)
 273 we show that when α decrease, the population density of the infected class reduce. From a biological point
 274 of view, we can say that biological memory has an impact on the density of both susceptible and infected
 275 classes.

276 6. Conclusion

277 The dynamics of a fractional-order SIS-epidemic model with intraspecific and interspecific competition
 278 have been studied. The validity of the model has been confirmed analytically by showing the existence,
 279 uniqueness, non-negativity, and boundedness of solutions. Three equilibrium points have been obtained
 280 namely the origin, the disease-free equilibrium point, and the endemic equilibrium point. Both origin and
 281 disease-free equilibrium points always exist while the endemic equilibrium point conditionally exists. The
 282 basic reproduction number \mathcal{R}_0 has been given which has a relationship with the local stability of the model.
 283 If $\mathcal{R}_0 < 1$ then the disease-free equilibrium point is locally asymptotically stable and if $\mathcal{R}_0 > 1$ then the
 284 disease-free equilibrium point losses its stability along with the existence of a locally asymptotically stable
 285 endemic equilibrium point. The global stability conditions of equilibrium points also have been found. The
 286 PRCC has been worked to investigate the most influential parameter. We have successfully shown that the
 287 infection rate and the death rate of the infected population due to interspecific competition becomes the
 288 most influential parameter for basic reproduction number and the population density of the infected class.



(a) Bifurcation diagram driven by ω_3 in interval $0 \leq \omega_3 \leq 1$



(b) Time-series for $\omega_3 = 0.2, 0.4, 0.6,$ and 0.8

Figure 8: Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to intraspecific competition (ω_3) with parameter values given by eq. (20)

289 We then investigate the impact of several parameters using numerical simulations including the infection
 290 rate, the recovery rate, the intraspecific competition, the interspecific competition, and the memory effect on
 291 the dynamics of the model. Bifurcation diagrams and time series have been given which show the existence
 292 of forward bifurcation, the decrease of susceptible and infected classes, and the decrease of convergence rate
 293 caused by the memory effect.

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 296 No. 023.17.2.677521/2021, under contract No. B/125/UN47.DI/PT.01.03/2022.

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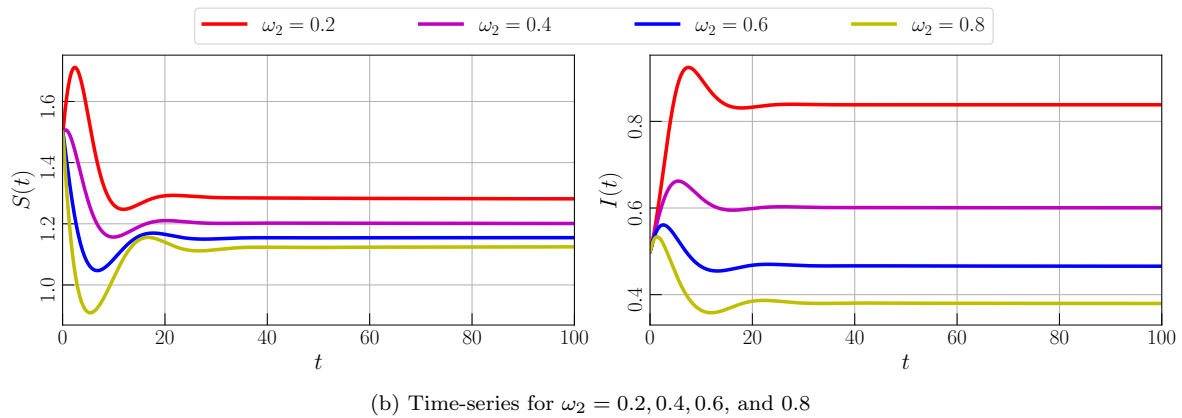
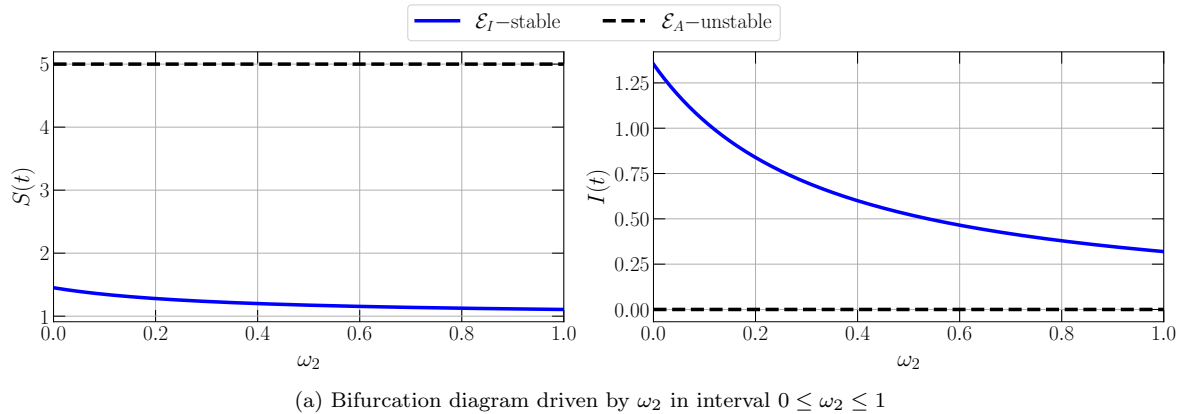


Figure 9: Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to interspecific competition (ω_2) with parameter values given by eq. (20)

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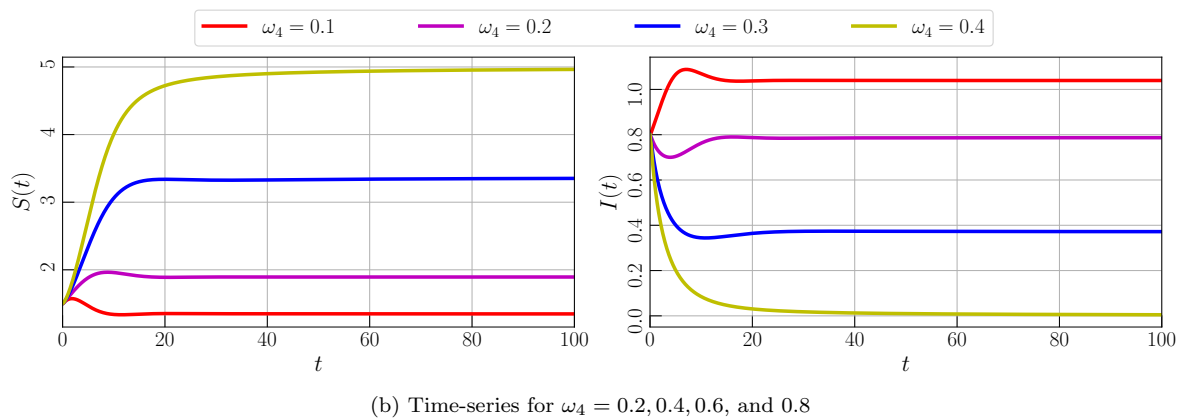
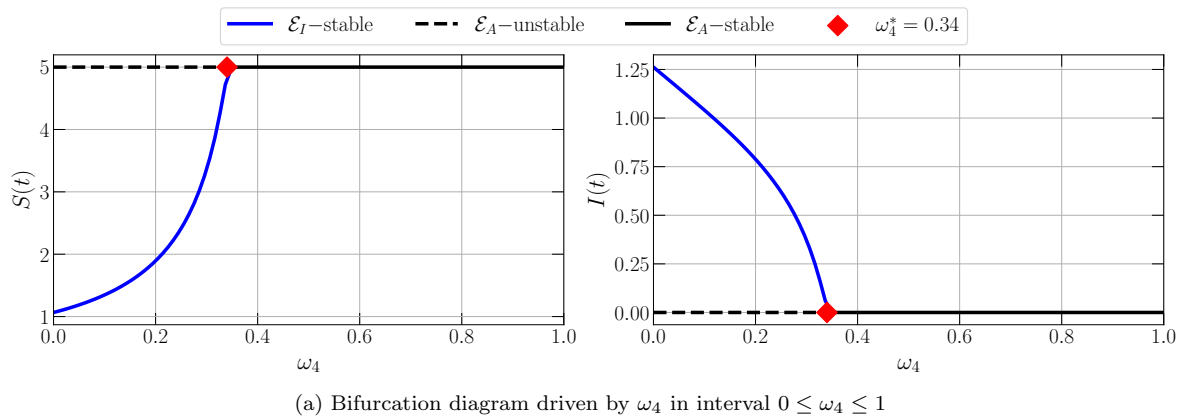


Figure 10: Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to interspecific competition (ω_4) with parameter values given by eq. (20)

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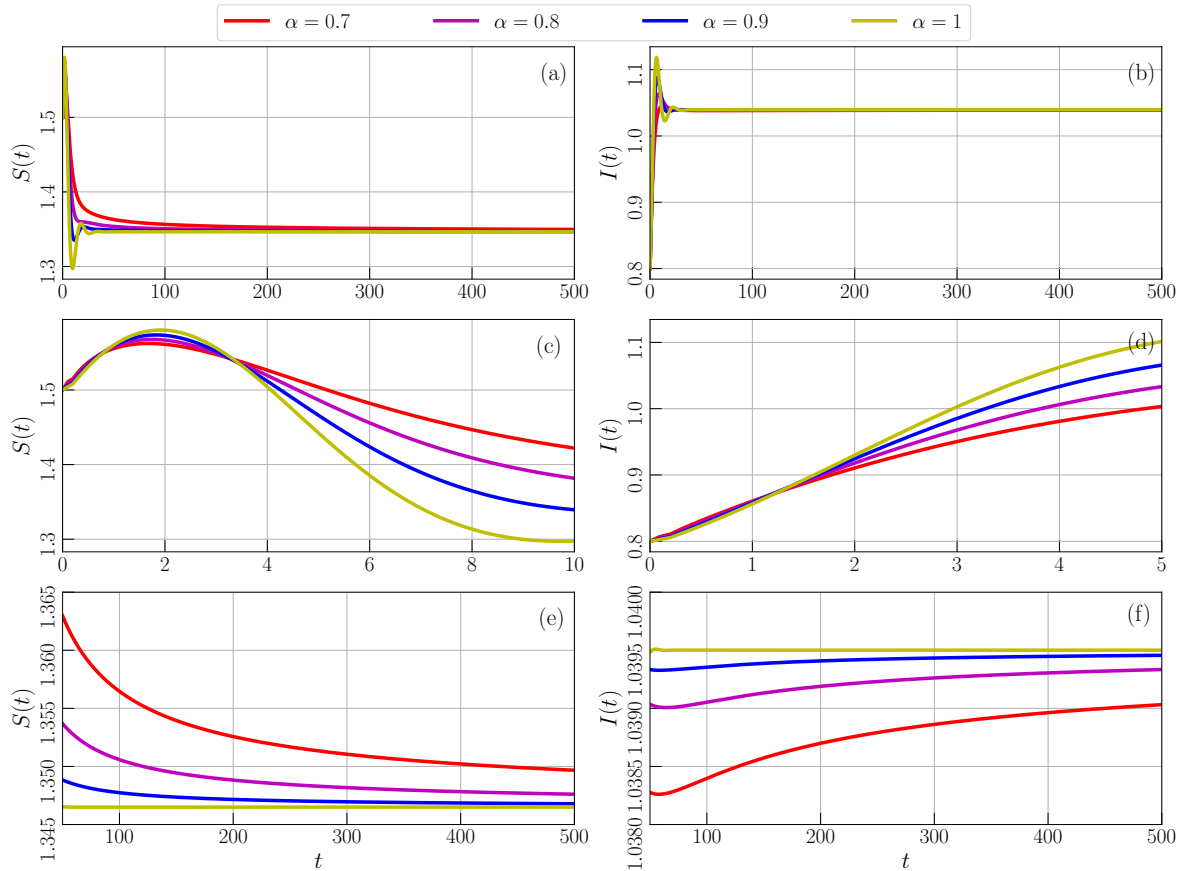


Figure 11: Time series of model (8) with parameter values given by eq. (20) for $\alpha = 0.7, 0.8, 0.9, 1$. **(a,b)** Time-series for $0 \leq t \leq 500$, **(c,d)** Local amplification of (a, b) around $0 \leq t \leq 10$, and **(e,f)** Local amplification of (a, b) around $100 \leq t \leq 500$

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DYNAMICS OF SIS–EPIDEMIC MODEL WITH COMPETITION INVOLVING FRACTIONAL-ORDER DERIVATIVE WITH POWER-LAW KERNEL

ISMAIL DJAKARIA^{1,*}, HASAN S. PANIGORO², EBENEZER BONYAH³, EMLI RAHMI², AND WAHAB MUSA⁴

¹Magister Mathematics Education Programme, Post-Graduate, Universitas Negeri Gorontalo,
Gorontalo 96128, Indonesia

²Biomathematics Research Group, Department of Mathematics, Faculty of Mathematics and Natural Sciences,
Universitas Negeri Gorontalo, Bone Bolango 96119, Indonesia

³Department of Mathematics Education, Akenten Appiah-Menka University of Skills Training and
Entrepreneurial Development, Kumasi 00233, Ghana

⁴Department of Electrical Engineering, Universitas Negeri Gorontalo, Gorontalo 96128, Indonesia

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Abstract. Infectious disease and competition play important roles in the dynamics of a population due to their capability to increase the mortality rate for each organism. In this paper, the dynamical behaviors of a single species population are studied by considering the existence of the infectious disease, intraspecific competition, and interspecific competition. The fractional-order derivative with a power-law kernel is utilized to involve the impact of the memory effect. The population is divided into two compartments namely the susceptible class and the infected class. The existence, uniqueness, non-negativity, and boundedness of the solution are investigated to confirm the biological validity. Three types of feasible equilibrium points are identified namely the origin, the disease-free, and the endemic points. All biological conditions which present the local and global stability are investigated. The global sensitivity analysis is given to investigate the most influential parameter to the basic reproduction number and the density of each class. Some numerical simulations including bifurcation diagrams and time series are also portrayed to explore more the dynamical behaviors.

Keywords: Infectious Disease; Competition; Fractional Derivative; Caputo Operator; Dynamical Behaviors

2010 AMS Subject Classification: 34A34, 92D30, 37N25, 37N30, 92B05.

*Corresponding author

E-mail address: iskar@ung.ac.id

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1. INTRODUCTION

The spread of infectious disease still becomes a fundamental issue not only because of the existence of the population but also to maintain the balance of biological systems. Several scientific methods are developed to discover better ways to suppress and control the rate of disease infection [1]. The preferred ways for the last decades for this epidemiological problems are given by mathematical approach using a deterministic model which is considered efficacious to understand the mechanisms of disease transmission and evaluate the appropriate control strategies [2–4]. The fundamental one which has become the basis of epidemiological modeling is given by [5] which develops the continuous-time deterministic model using first-order derivative as the operator. This model is successfully developed in couple of ways such as the continuous-time single species epidemiological modeling with first-order derivative [6–9], the discrete-time single species epidemiological modeling [10–12], the stochastic single-species epidemiological modeling [13, 14], and the continuous-time eco-epidemiological modeling [15–17].

Apart from those operators, several researchers prefer to use the fractional-order derivative to accomplish their problems the biological modeling. See [18–20] and references therein for some examples in epidemiological modeling. The fractional-order derivative is chosen by considering the capability of this operator to describe the current state of the biological object as the impact of all of its previous conditions which are known as the memory effect [21, 22]. In the epidemiological model, the transmission of disease may slow down and be forestalled by the susceptible population as the impact of the memory [23]. Some fractional-order derivative has been developed and successfully applied in epidemiological modeling such as the Riemann-Liouville, Caputo, Caputo-Fabrizio, and Atangana-Baleanu [24–27]. From all of the given operators, the Caputo fractional-order derivative has the complete tools for dynamical analysis such as the existence and uniqueness, non-negativity and boundedness, local dynamics, global dynamics, and some bifurcation analysis. Consequently, the Caputo operator will be used in this paper where defined later in the next section.

In this work, we develop the epidemiological model based on the SIR model given by [5]. For single-species conditions, this model is only popular for the infectious diseases that appeared in the human population. In facts, infectious diseases also threaten the existence of the animal population which disturbs the balance of the ecosystem. For examples, the infectious diseases in endemic species such as Orangutans [28], Tarsius [29], Sumatran Tiger [30], and Komodo dragon [31]. Moreover, the natural behaviors of animals that endanger the existence of their populations are the intraspecific competition among them to preserve their food sources [32–34]. For these reasons, developing and investigating the dynamics of the epidemiological model by considering the impact of intraspecific competition and the memory effect are critical issues that become the novelty of our research.

The whole of this paper is organized in the following procedure: In Section 2, the mathematical modeling consists of model formulation, existence, uniqueness, non-negativity, and boundedness are given. The analytical results including the existence of equilibrium points and their local and global dynamics are completely investigated in Section 3. To show the most influential parameter of the model, the global sensitivity analysis is provided

by Section 4. Some numerical simulations as well as bifurcation diagrams and time-series are presented in Section 5 to explore more about the dynamical behaviors of the model. This work ends by giving a conclusion in Section 6.

2. MATHEMATICAL MODELING

This section studies about mathematical modeling consisting of the model formulation, existence, uniqueness, non-negativity, and boundedness of solution. The mathematical model is constructed by a deterministic approach using a differential equation. We first give some assumptions to restrain the model so it does not get too complicated. We next interpret the giving assumptions to the mathematical formula using the first-order derivative as the operator. A diagram is presented to show the impact of each assumption on the flow of population density for each compartment. To involve the impact of the memory effect, the Caputo fractional-order derivative is applied to the model. For the mathematical model's validity, we show that the solution of the model always exists, unique, non-negative, and bounded.

2.1. Model Formulation. In this work, the model is constructed from a single population growth model. We first assume there exists a population in a habitat that grows proportionally to its density and bounded due to the intraspecific competition. Let $N(t)$ be the population at time t , r is the birth rate, μ is the natural death rate, and ω is the death rate as a result of competition. Thus, we have a first-order differential equation as follows.

$$(1) \quad \frac{dN}{dt} = (r - \mu)N - \omega N^2.$$

Next, we assume that the population is exposed by infectious disease. The population N is divided into two compartments namely the susceptible class (S) and infected class (I) where $N = S + I$. The susceptible class is infected by disease bilinearly with infection rate β . The competition is divided into two cases namely the intraspecific competition for each susceptible and infected class, and the interspecific competition between susceptible and infected classes. As result, the following model is received.

$$(2) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - \mu I, \end{aligned}$$

where ω_i , $i = 1, 2$ respectively denote the death rate of the susceptible population as the results of intraspecific and interspecific competitions between susceptible and susceptible classes, and susceptible and infected classes. The parameters ω_i , $i = 3, 4$ denote the death rate of the infected population as the result of competition between infected and infected classes, and susceptible and infected classes. In our works, we also assume that each organism has the capability to survive the disease. Thus, we define η as the recovery rate. Since each organism that survives from the disease has a chance to be re-infected, this type of population will be again susceptible. Finally, we have

a mathematical model as follows.

$$(3) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

All of the given assumptions and their mathematical modeling are described in Figure 1.

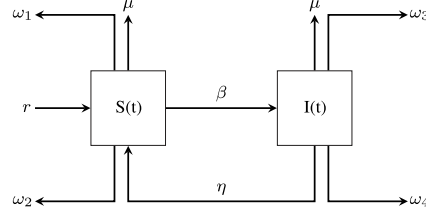


FIGURE 1. Compartment diagram of model (3)

Now, the Caputo fractional-order derivative will be applied in order to conduct the impact of the memory effect on the population growth rate. The similar procedure is adopted from [35]. The first-order derivatives on the left-hand side of model (3) are replaced by the Caputo fractional-order derivative defined as follows.

Definition 1. [36] Suppose $0 < \alpha \leq 1$. The Caputo fractional derivative of order $-\alpha$ is defined by

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

where $t \geq 0$, $f \in C^n([0, +\infty), \mathbb{R})$, and Γ is the Gamma function.

Applying Definition 1 to eq. (3), the following model is obtained.

$$(5) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

Since the given process above makes the dimension of time at the left-hand side become t^α , some parameters need to be rescaled so that there are no differences between the time's dimensions at the left-hand side with the right-hand side of model (5). By applying time rescale to some parameters, we have the model as follows.

$$(6) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r^\alpha - \mu^\alpha)S - \omega_1^\alpha S^2 - (\omega_2^\alpha + \beta^\alpha)SI + \eta^\alpha I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta^\alpha - \omega_4^\alpha)SI - \omega_3^\alpha I^2 - (\eta^\alpha + \mu^\alpha)I. \end{aligned}$$

Let $r^\alpha = \hat{r}$, $\mu^\alpha = \hat{\mu}$, $\omega_1^\alpha = \hat{\omega}_1$, $\omega_2^\alpha = \hat{\omega}_2$, $\omega_3^\alpha = \hat{\omega}_3$, $\omega_4^\alpha = \hat{\omega}_4$, $\beta^\alpha = \hat{\beta}$, and $\eta^\alpha = \hat{\eta}$. Thus, we acquire

$$(7) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (\hat{r} - \hat{\mu})S - \hat{\omega}_1 S^2 - (\hat{\omega}_2 + \hat{\beta})SI + \hat{\eta}I, \\ {}^C \mathcal{D}_t^\alpha I &= (\hat{\beta} - \hat{\omega}_4)SI - \hat{\omega}_3 I^2 - (\hat{\eta} + \hat{\mu})I. \end{aligned}$$

For simplicity, by dropping $\hat{\cdot}$ for each parameter, we obtain the final model as follows.

$$(8) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I = F_1(N(t)), \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I = F_2(N(t)). \end{aligned}$$

Equation (8) is the final proposed model in this paper. Although model (8) seems classic and simple, this model will be powerful to solve and investigate the existence of a closed population in a certain area without any outside intervention. Our literature review also shows that the model (8) has heretofore never been studied. Now, the basic properties of model (8) such as the existence uniqueness, non-negativity, and boundedness are investigated to confirm its biological validity.

2.2. Existence and Uniqueness. In this subsection, we will show that the model (8) has a unique solution. A similar manner given by [37] is used. Thus, the following theorem is presented to show the existence and uniqueness of the solution of model (8).

Theorem 1. *The model (8) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ has a unique solution.*

Proof. Consider model (8) with positive initial condition with $F : [0, \infty) \rightarrow \mathbb{R}^2$ where $F(N) = (F_1(N), F_2(N))$, $N \equiv N(t)$ and $\theta \equiv \{(S, I) \in \mathbb{R}_+^2 : \max\{|S|, |I|\} \leq M\}$ for sufficiently large M . Then, for any $N = (S, I)$ and $\bar{N} = (\bar{S}, \bar{I})$, $N, \bar{N} \in \theta$, we have

$$\begin{aligned} \|F(N) - F(\bar{N})\| &= |F_1(N) - F_1(\bar{N})| + |F_2(N) - F_2(\bar{N})| \\ &= \left| [(r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I] - [(r - \mu)\bar{S} - \omega_1 \bar{S}^2 - (\omega_2 + \beta)\bar{S}\bar{I} + \eta \bar{I}] \right| + \\ &\quad \left| [(\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I] - [(\beta - \omega_4)\bar{S}\bar{I} - \omega_3 \bar{I}^2 - (\eta + \mu)\bar{I}] \right| \\ &\leq (r + \mu) |S - \bar{S}| + \omega_1 |S^2 - \bar{S}^2| + (\omega_2 + \beta) |SI - \bar{S}\bar{I}| + \eta |I - \bar{I}| + (\beta + \omega_4) |SI - \bar{S}\bar{I}| \\ &\quad + \omega_3 |I^2 - \bar{I}^2| + (\eta + \mu) |I - \bar{I}| \\ &= (r + \mu) |S - \bar{S}| + \omega_1 |(S + \bar{S})(S - \bar{S})| + (\omega_2 + \omega_4 + 2\beta) |I(S - \bar{S}) + \bar{S}(I - \bar{I})| \\ &\quad + (2\eta + \mu) |I - \bar{I}| + \omega_3 |(I + \bar{I})(I - \bar{I})| \\ &\leq (r + \mu) |S - \bar{S}| + 2\omega_1 M |S - \bar{S}| + (\omega_2 + \omega_4 + 2\beta) M |S - \bar{S}| \\ &\quad + (\omega_2 + \omega_4 + 2\beta) M |I - \bar{I}| + (2\eta + \mu) |I - \bar{I}| + 2\omega_3 M |I - \bar{I}| \\ &= [(r + \mu) + 2\omega_1 M + (\omega_2 + \omega_4 + 2\beta) M] |S - \bar{S}| + [(\omega_2 + \omega_4 + 2\beta) M + (2\eta + \mu) + 2\omega_3 M] |I - \bar{I}| \\ &\leq L \|N - \bar{N}\|, \end{aligned}$$

where $L = (\omega_2 + \omega_4 + 2\beta)M + \mu + \max\{r + 2\omega_1 M, 2(\eta + \omega_3 M)\}$. Therefore, $F(N)$ satisfies the Lipschitz condition. Obeying Lemma 5 in [38], we conclude that model (8) with positive initial condition has a unique solution. \square

2.3. Non-negativity and Boundedness. The non-negativity and boundedness properties of the solutions of the model (8) are given in the following theorem.

Theorem 2. *All solution of the model (8), which start in $\mathbb{R}_+^2 := \{(S, I) \mid S \geq 0, I \geq 0, (S, I) \in \mathbb{R}^2\}$ are uniformly bounded and non-negative.*

Proof. To prove the boundedness of the solutions of the model (8), the same approach of [38] is adopted. Let consider the function $N = S + I$. Then,

$$\begin{aligned} {}^C \mathcal{D}_t^\alpha N &= {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\ &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I + (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I \\ &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I. \end{aligned}$$

Hence, for each $\mu > 0$,

$$\begin{aligned} {}^C \mathcal{D}_t^\alpha N + \mu N &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I + \mu S + \mu I \\ &= rS - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\ &= -\omega_1 \left(S - \frac{r}{2\omega_1} \right)^2 + \frac{r^2}{4\omega_1} - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\ &\leq \frac{r^2}{4\omega_1} \end{aligned}$$

By using the comparison theorem in [39], we obtain $N(t) \leq N(0)E_\alpha(-\mu t^\alpha) + \frac{r^2}{4\omega_1} t^\alpha E_{\alpha, \alpha+1}(-\mu t^\alpha)$, where E_α and $E_{\alpha, \alpha+1}$ is the Mittag-Leffler function with one and two parameters. According to Lemma 5 and Corollary 6 in [39], we have $N(t) \leq \frac{r^2}{4\mu\omega_1}$, as $t \rightarrow \infty$. Therefore, all solutions of model (8) starting in \mathbb{R}_+^2 are uniformly bounded in the region Φ , where $\Phi = \left\{ (S, I) \in \mathbb{R}_+^2 : S + I \leq \frac{r^2}{4\mu\omega_1} + \varepsilon, \varepsilon > 0 \right\}$. Next, we prove that all solutions of model (8) are non-negative. By model (8), we have ${}^C \mathcal{D}_t^\alpha S|_{S=0} = \eta I \geq 0$ and ${}^C \mathcal{D}_t^\alpha I|_{I=0} = 0 \geq 0$. Based on Lemmas 5 and 6 in [40], we conclude that the solutions of model (8) are non-negative. \square

3. ANALYTICAL RESULTS

In this section, the dynamics of model (8) are shown analytically including the existence of equilibrium points, and their local and global stability.

3.1. Existence of Equilibrium Points. To find the equilibrium points of model (8), we must have

$$(9) \quad [(r - \mu) - \omega_1 S - (\omega_2 + \beta)I]S + \eta I = 0,$$

$$(10) \quad [(\beta - \omega_4)S - \omega_3 I - (\eta + \mu)]I = 0.$$

If $I = 0$ is substituted to (9), we obtain

$$(11) \quad [(r - \mu) - \omega_1 S] S = 0.$$

From eq. (11), we get $S = 0$ and $S = \frac{r - \mu}{\omega_1}$. Thus, we have two equilibrium points here namely $\mathcal{E}_0 = (0, 0)$, and $\mathcal{E}_A = \left(\frac{r - \mu}{\omega_1}, 0\right)$. The equilibrium point \mathcal{E}_0 is called the origin point which represents the extinction of both susceptible and infected populations. Since $\mathcal{E}_0 \in \mathbb{R}_+^2$, this equilibrium point always exists. Furthermore, the equilibrium point \mathcal{E}_A is called the disease-free equilibrium point (DFEP) which describes the condition where the infectious disease does not exist anymore in the population. According to the biological condition, it is natural that the birth rate r is greater than its death rate μ . By assuming $r > \mu$, the origin point $\mathcal{E}_A \in \mathbb{R}_+^2$ also always exists. By simple calculation, we also obtain the basic reproduction number \mathcal{R}_0 given by

$$(12) \quad \mathcal{R}_0 = \frac{(r - \mu)\beta}{(r - \mu)\omega_4 + (\eta + \mu)\omega_1}.$$

The basic reproduction number is utilized to show the dynamical behavior of each equilibrium point and to describe whether the infectious disease becomes endemic or not. Since $r > \mu$, the value of \mathcal{R}_0 is always positive. Now, let's concern the eq. (9) and (10). By solving eq. (10), we attain

$$(13) \quad S = \frac{\omega_3 I + (\eta + \mu)}{\beta - \omega_4}.$$

If we substitute eq. (13) to (9), the following polynomial equation holds.

$$(14) \quad k_1 I^2 + k_2 I + k_3 = 0,$$

where

$$\begin{aligned} k_1 &= ((\beta - \omega_4)(\beta + \omega_2) + \omega_1 \omega_3) \omega_3, \\ k_2 &= (\beta - \omega_4)((\beta + \omega_2)\mu + (\omega_2 + \omega_4)\eta - (r - \mu)\omega_3) + 2(\eta + \mu)\omega_1 \omega_3, \\ k_3 &= \frac{(1 - \mathcal{R}_0)(r - \mu)(\eta + \mu)\beta}{\mathcal{R}_0}. \end{aligned}$$

Therefore, we acquire the endemic point (EEP)

$$(15) \quad \mathcal{E}_I = \left(\frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}, \bar{\gamma} \right),$$

where $\bar{\gamma}$ is the positive root of polynomial equation (14). From (15), we find that $\beta > \omega_4$ must be fulfilled so that $\mathcal{E}_I \in \mathbb{R}_+^2$. Moreover, EEP exists if $\bar{\gamma} > 0$. From eq. (14), we have k_1 is always positive. Thus, the value of the $\bar{\gamma}$ depends on k_2 and k_3 . Furthermore, eq. (14) has real number roots if $k_2^2 \geq 4k_1 k_3$. By applying simple algebra, if $k_3 > 0$ and $k_2 < 0$ then we have two positive roots of eq. (14), if $k_3 > 0$ and $k_2 > 0$ then we do not have any positive roots of eq. (14), and if $k_3 < 0$ then we have a positive root of eq. (14). Finally, we have the following theorem.

Theorem 3. *Let $\beta > \omega_4$. The existence of EEP \mathcal{E}_1 is shown by the following statement.*

- (i) *If $k_2^2 < 4k_1k_3$ then \mathcal{E}_1 does not exist.*
- (ii) *If $k_2^2 = 4k_1k_3$ and*
 - (ii.i) *if $k_2 > 0$ then \mathcal{E}_1 does not exist.*
 - (ii.ii) *if $k_2 < 0$ then \mathcal{E}_1 exists and unique.*
- (iii) *If $k_2^2 > 4k_1k_3$ and*
 - (iii.i) *if $k_3 > 0$ and $k_2 < 0$ then we have a pair of \mathcal{E}_1 .*
 - (iii.ii) *if $k_3 > 0$ and $k_2 > 0$ then \mathcal{E}_1 does not exist.*
 - (iii.iii) *if $k_3 < 0$ then \mathcal{E}_1 exists and unique.*

Denote that $k_2^2 > 4k_1k_3$ is always satisfied and $k_3 < 0$ for $\mathcal{R}_0 > 1$, then the following lemma holds.

Lemma 4. *EEP \mathcal{E}_1 exists and unique if $\mathcal{R}_0 > 1$.*

3.2. Local Dynamics. The local dynamics of model (8) are obtained by applying the Matignon condition which is defined as follows.

Theorem 5. *[Matignon condition [36]] An equilibrium point \bar{x}^* is locally asymptotically stable (LAS) if all eigenvalues λ_j of the Jacobian matrix $J = \frac{\partial \vec{f}}{\partial \vec{x}}$ at \bar{x}^* satisfy $|\arg(\lambda_j)| > \frac{\alpha\pi}{2}$. If there exists at least one eigenvalue satisfy $|\arg(\lambda_k)| > \frac{\alpha\pi}{2}$ while $|\arg(\lambda_l)| < \frac{\alpha\pi}{2}$, $k \neq l$, then \bar{x}^* is a saddle-point.*

Therefore, to study the local dynamics of model (8), we first compute its Jacobian matrix at the point (S, I) which gives

$$(16) \quad \mathcal{J}(S, I) = \begin{bmatrix} (r - \mu) - 2\omega_1 S - (\omega_2 + \beta)I & -(\omega_2 + \beta)S + \eta \\ (\beta - \omega_4)I & (\beta - \omega_4)S - 2\omega_3 I - (\eta + \mu) \end{bmatrix}.$$

Obeying Theorem 5 and using Jacobian matrix (16), we discuss the local stability for each equilibrium point in the next subsection.

3.3. Dynamical behavior around \mathcal{E}_0 . LAS condition of \mathcal{E}_0 is obtained by identifying the eigenvalues of the Jacobian matrix (16) at the point $(S, I) = (0, 0)$. We receive

$$\mathcal{J}(S, I)|_{\mathcal{E}_0} = \begin{bmatrix} r - \mu & \eta \\ 0 & -(\eta + \mu) \end{bmatrix}.$$

Therefore, we have $\lambda_1 = r - \mu$ and $\lambda_2 = -(\eta + \mu)$. Since $r > \mu$ and $\lambda_2 < 0$, we have $|\arg(\lambda_1)| = 0 < \frac{\alpha\pi}{2}$ and $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$. According to Theorem 5, the following theorem holds.

Theorem 6. *The origin point \mathcal{E}_0 is always a saddle point.*

3.4. Dynamical behavior around \mathcal{E}_A . For $(x, y) = \left(\frac{r-\mu}{\omega_1}, 0\right)$, the Jacobian matrix (16) becomes

$$\mathcal{J}(S, I)|_{\mathcal{E}_A} = \begin{bmatrix} -(r-\mu) & \eta - \frac{(\omega_2+\beta)(r-\mu)}{\omega_1} \\ 0 & \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0} \end{bmatrix},$$

which gives a pair of eigenvalues $\lambda_1 = -(r-\mu)$ and $\lambda_2 = \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0}$. Denote $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$ as the impact of $\lambda_1 < 0$. Hence, the sign of λ_2 takes the role in describing local dynamics around \mathcal{E}_A . To obtain $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$, we need $\lambda_2 < 0$ which is fulfilled if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ then $|\arg(\lambda_2)| = 0 < \frac{\alpha\pi}{2}$. Following the Matignon condition given in Theorem 5, the following theorem is successfully attained.

Theorem 7. *If $\mathcal{R}_0 < 1$ then \mathcal{E}_A is LAS and a saddle point if $\mathcal{R}_0 > 1$.*

3.5. Dynamical behavior around \mathcal{E}_I . To identify the local stability of \mathcal{E}_I , we first compute the Jacobian matrix (16) evaluated at \mathcal{E}_I . We generate

$$(17) \quad \mathcal{J}(S, I)|_{\mathcal{E}_I} = \begin{bmatrix} -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu}\right] & -\frac{(\omega_2+\beta)(\omega_3\bar{\gamma}+\eta+\mu)}{\beta-\omega_4} + \eta \\ (\beta-\omega_4)\bar{\gamma} & -\omega_3\bar{\gamma} \end{bmatrix}.$$

The eigenvalues of (17) are given by $\lambda_1 = \frac{1}{2} \left(\xi_1 + \sqrt{\xi_1^2 - 4\xi_2} \right)$ and $\lambda_2 = \frac{1}{2} \left(\xi_1 - \sqrt{\xi_1^2 - 4\xi_2} \right)$ where

$$\begin{aligned} \xi_1 &= -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu} + \omega_3\bar{\gamma}\right], \\ \xi_2 &= \left[\left(\frac{\omega_1\omega_3}{\beta-\omega_4} + \omega_2 + \beta\right)(\omega_3\bar{\gamma}+\eta+\mu) + \left(\frac{\omega_3\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu} + 1\right)(\beta-\omega_4)\eta\right]\bar{\gamma}. \end{aligned}$$

It is easy to proof that $\xi_1 < 0$ and $\xi_2 > 0$ since $\beta > \omega_4$ becomes the existence condition. As the impact, $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$, $i = 1, 2$ and hence the LAS always hold for EEP. Thus, the following theorem holds.

Theorem 8. *EEP \mathcal{E}_I is always LAS.*

3.6. Global Dynamics. In this subsection, the global dynamics of model (8) are studied. The biological conditions of equilibrium points are investigated so that those points are globally asymptotically stable (GAS). Since the origin is always a saddle point, we focus on studying GAS conditions for DFEP and EEP. The next two theorems are given for the global dynamics.

Theorem 9. *DFEP \mathcal{E}_A is GAS if $\omega_1 > \frac{(\omega_2+\beta)r}{\mu}$.*

Proof. We define a positive Lyapunov function as follows.

$$(18) \quad \mathcal{V}_A(S, I) = \left(S - \frac{r-\mu}{\omega_1} - \frac{r-\mu}{\omega_1} \ln \frac{\omega_1 S}{r-\mu} \right) + I.$$

If we calculate the Caputo fractional derivative of $\mathcal{V}_A(S, I)$ along the solution of model (8) and use Lemma 3.1 in [41], we get

$${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = \left(\frac{S - \frac{r-\mu}{\omega_1}}{S} \right) {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I$$

$$\begin{aligned}
&= -\omega_1 \left(S - \frac{r-\mu}{\omega_1} \right)^2 + \frac{(r-\mu)(\omega_2+\beta)I}{\omega_1} - \frac{(r-\mu)\eta I}{\omega_1 S} - (\omega_2+\omega_4)SI - \omega_3 I^2 - \mu I \\
&\leq -\omega_1 \left(S - \frac{r-\mu}{\omega_1} \right)^2 - \left(\mu - \frac{(\omega_2+\beta)r}{\omega_1} \right) I
\end{aligned}$$

Since $\omega_1 > \frac{(\omega_2+\beta)r}{\mu}$, we have ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$, and ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$ only when $(S, I) = \left(\frac{r-\mu}{\omega_1}, 0 \right)$. This means that the singleton $\{\mathcal{E}_A\}$ is the only invariant set where ${}^C\mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$. By Lemma 4.6 in [42], we can conclude that every solution of model (8) tends to DFEP \mathcal{E}_A . \square

Theorem 10. *EEP \mathcal{E}_I is GAS if $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$.*

Proof. We first define $\vartheta = \frac{\omega_3\bar{\gamma}+(\eta+\mu)}{\beta-\omega_4}$ and hence $\mathcal{E}_I = (\vartheta, \bar{\gamma})$. Now, a positive Lyapunov function is presented as follows.

$$(19) \quad \mathcal{V}_I(S, I) = \left(S - \vartheta - \vartheta \ln \frac{S}{\vartheta} \right) + \left(I - \bar{\gamma} - \bar{\gamma} \ln \frac{I}{\bar{\gamma}} \right)$$

Following Lemma 3.1 in [41], we reach

$$\begin{aligned}
{}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) &= \left(\frac{S-\vartheta}{S} \right) {}^C\mathcal{D}_t^\alpha S + \left(\frac{I-\bar{\gamma}}{I} \right) {}^C\mathcal{D}_t^\alpha I \\
&= (S-S^*) \left((r-\mu) - \omega_1 S - (\omega_2+\beta)I + \frac{\eta I}{S} \right) + (I-\bar{\gamma}) \left((\beta-\omega_4)S - \omega_3 I - (\eta+\mu) \right) \\
&= -\omega_1 (S-\vartheta)^2 - \omega_3 (I-\bar{\gamma})^2 - (\omega_2+\omega_4)(S-S^*)(I-\bar{\gamma}) \\
&\leq -\left(\omega_1 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} \right) \right) (S-\vartheta)^2 - \left(\omega_3 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} \right) \right) (I-\bar{\gamma})^2
\end{aligned}$$

Denote that ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$ as a result of $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$. We also have that ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$ only when $(S, I) = (\vartheta, \bar{\gamma})$. Therefore, the singleton $\{\mathcal{E}_I\}$ is the only invariant set where ${}^C\mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$. Obeying Lemma 4.6 in [42], every solution of model (8) tends to EEP \mathcal{E}_I . \square

4. GLOBAL SENSITIVITY ANALYSIS

In this section, the global sensitivity analysis is studied to investigate the most influential parameters of model (8). Global sensitivity analysis is calculated using Partial Rank Coefficient Correlation (PRCC) [43], where the random data processed in PRCC is generated using Saltelli sampling [44]. Two biological components become the objective function for the PRCC namely the basic reproduction number (\mathcal{R}_0) and the population density of infected class ($I(t)$). We first investigate the most influential parameter to the basic reproduction number (\mathcal{R}_0). From eq. (12), we acquire that only r , μ , ω_1 , ω_4 , and η have the influence on the value of \mathcal{R}_0 . The birth rate and the natural death rate also can be fixed since some cases in the epidemiological model has the values of these parameters. Thus, only β , η , ω_1 , and ω_4 will be computed for PRCC. The Figure 2 is given for the results. We

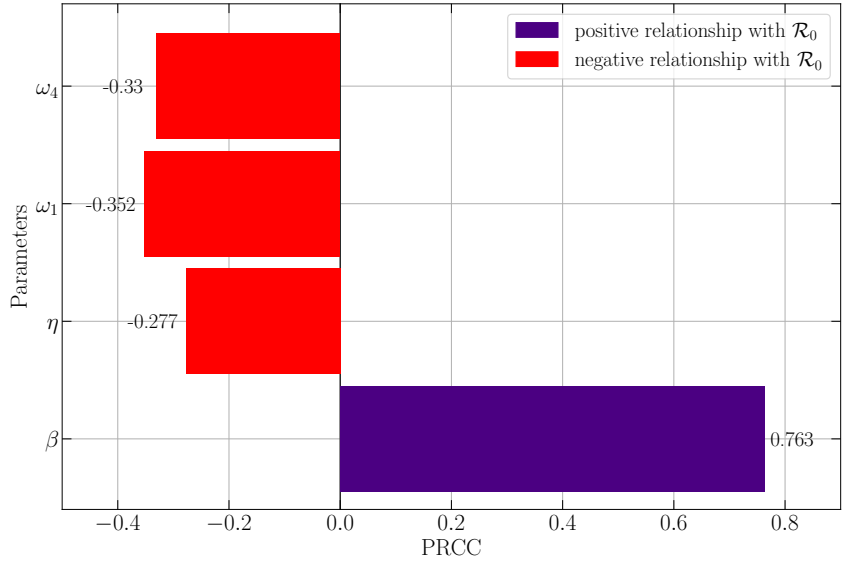
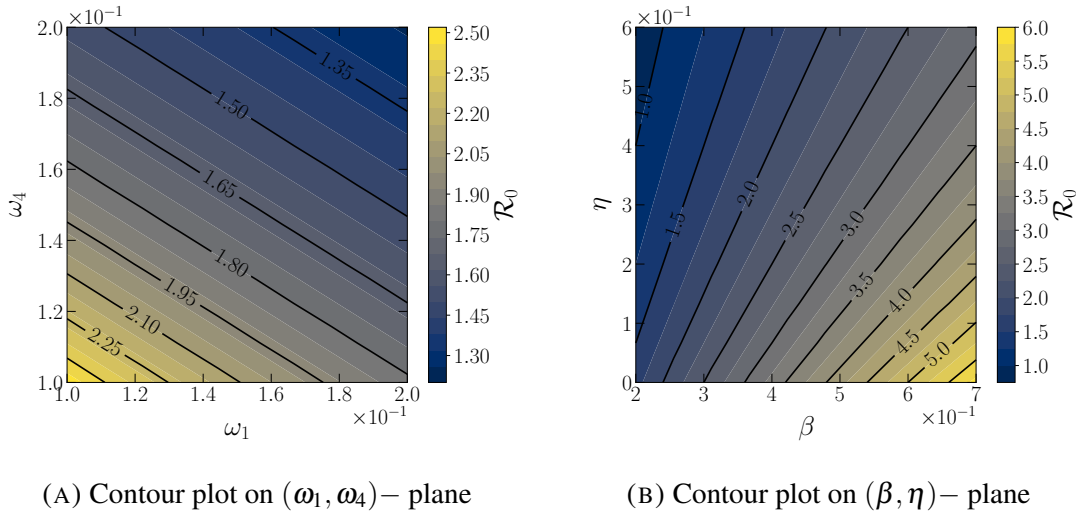


FIGURE 2. PRCC results for the parameters of \mathcal{R}_0



(A) Contour plot on (ω_1, ω_4) – plane

(B) Contour plot on (β, η) – plane

FIGURE 3. Contour plots for the parameters respect to \mathcal{R}_0

have $\beta = 0.763$, $\omega_1 = -0.352$, $\omega_4 = -0.33$, and $\eta = -0.277$ as the coefficient correlation such that the infection rate (β) becomes the most influential parameter to \mathcal{R}_0 and followed by ω_1 , ω_4 , and η , respectively. It shows that the infection rate (β) as the most influential parameter has a positive relationship with the basic reproduction number (\mathcal{R}_0) which means that \mathcal{R}_0 will significantly increases when β increases. The rest ω_1 , ω_4 , and η have a negative relationship with \mathcal{R}_0 which means that by reducing the value of those parameters, the basic reproduction number (\mathcal{R}_0) will increases. To show the impact of these parameters on \mathcal{R}_0 , the contour plots are also portrayed in Figure 3.

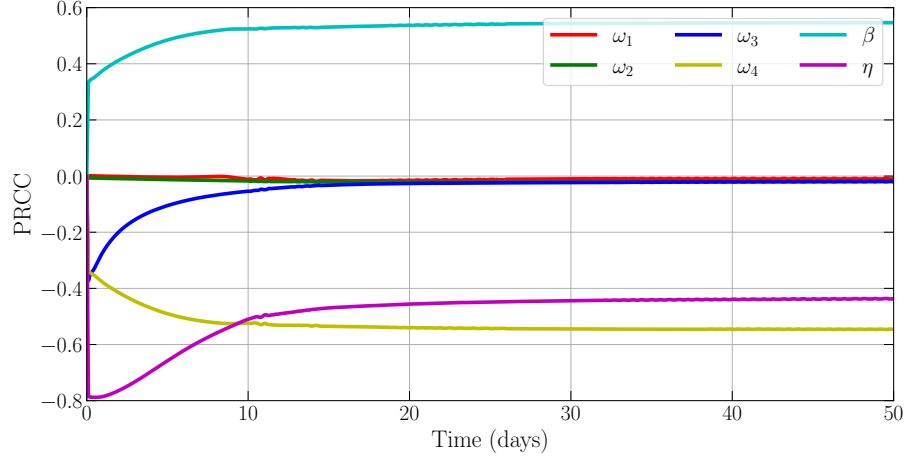
FIGURE 4. PRCC results for the parameters of $I(t)$

TABLE 1. PRCC results in respect to the population density of infected class

Parameter	Description	PRCC	Rank	Relationship with $I(t)$
ω_1	The death rate of susceptible population due to the intraspecific competition	-0.00851	6	Negative relationship
ω_2	The death rate of susceptible population due to the interspecific competition	-0.01938	5	Negative relationship
ω_3	The death rate of infected population due to the intraspecific competition	-0.01990	4	Negative relationship
ω_4	The death rate of infected population due to the interspecific competition	-0.54635	1	Negative relationship
β	The infection rate	0.54631	2	Positive relationship
η	The recovery rate	-0.43606	3	Negative relationship

Next, we identify the most influential parameter to the population density of infected class ($I(t)$). Quite similar to previous work, the value of r and μ are fixed but the rest of the parameters are involved to compute PRCC. PRCC values are computed for $0 \leq t \leq 50$ which is considered sufficient enough to see the convergence for each parameter through the PRCC. We portray the PRCC results in Figure 4 while the PRCC values, ranks, and the relationship between each parameter and $I(t)$ are given in Table 1. From those simulations, we conclude that the death rate of infected population due to interspecific competition between susceptible and infected classes (ω_4) become the most influential parameter to the population density ($I(t)$) followed respectively by β , η , ω_3 , ω_2 , and ω_1 . In the next section, the numerical simulations including bifurcation diagram and time-series are presented to

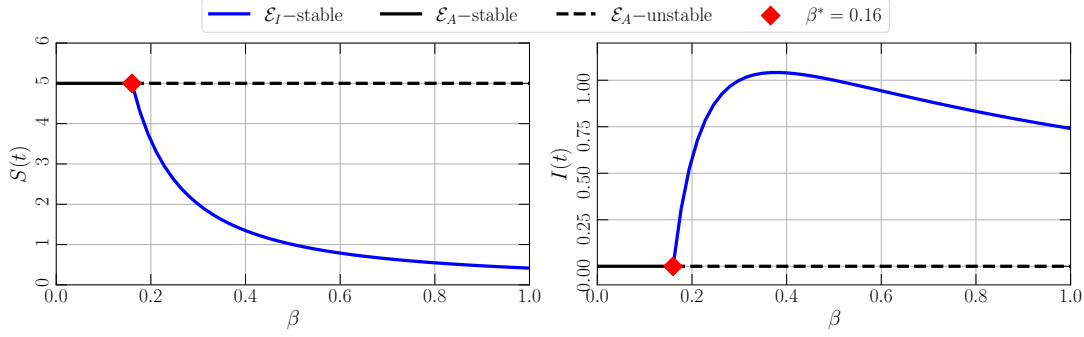
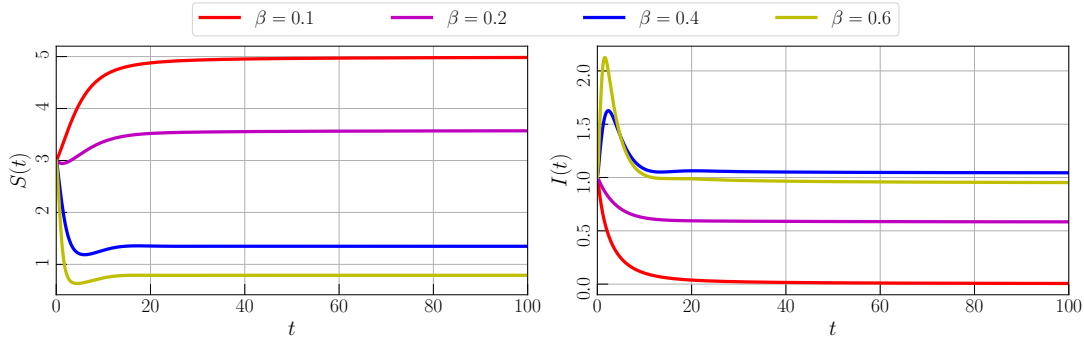

 (A) Bifurcation diagram driven by β in interval $0 \leq \beta \leq 1$

 (B) Time-series for $\beta = 0.1, 0.2, 0.4,$ and 0.6

 FIGURE 5. Bifurcation diagram and times-series of model (8) driven by the infection rate (β) with parameter values given by eq. (20)

show the impact of the infection rate (β), recovery rate (η), intraspecific competition (ω_1 and ω_3), and interspecific competition (ω_2 and ω_4) to the dynamical behaviors of model (8).

5. NUMERICAL SIMULATIONS

In this section, the dynamical behaviors of model (8) including bifurcation diagram and time-series are studied numerically. To obtain the bifurcation diagram and the corresponding time-series of model (8), the predictor-corrector scheme developed by Diethelm et al. is employed [45]. Since the model does not investigate a specific epidemiological case, we use hypothetical parameters for all numerical simulations. we set the parameter values as follows.

$$(20) \quad r = 0.6, \mu = 0.1, \omega_1 = 0.1, \omega_2 = 0.1, \omega_3 = 0.1, \omega_4 = 0.1, \beta = 0.4, \eta = 0.2, \text{ and } \alpha = 0.9$$

We start our work by investigating the impact of infection rate (β) on the dynamics of model (8). The value of β is varied in the interval $0 \leq \beta \leq 1$ and we then compute the numerical solutions. To obtain the bifurcation diagram, we plot the tail of solutions for each β together with the LAS condition of \mathcal{E}_A . As result, we obtain a

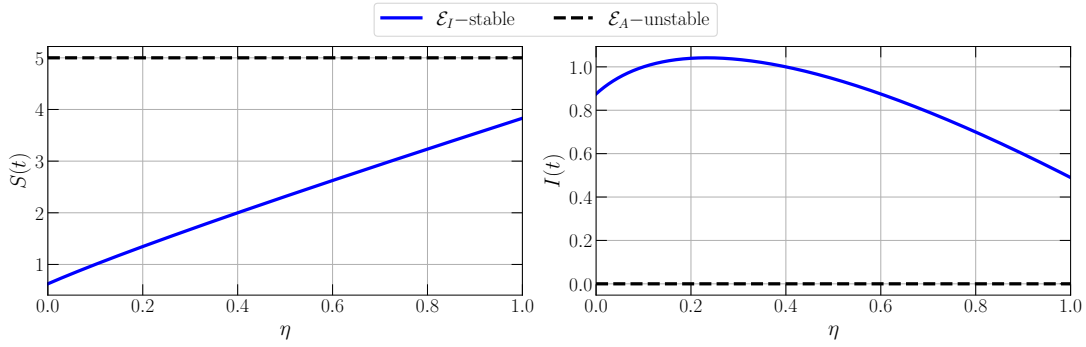
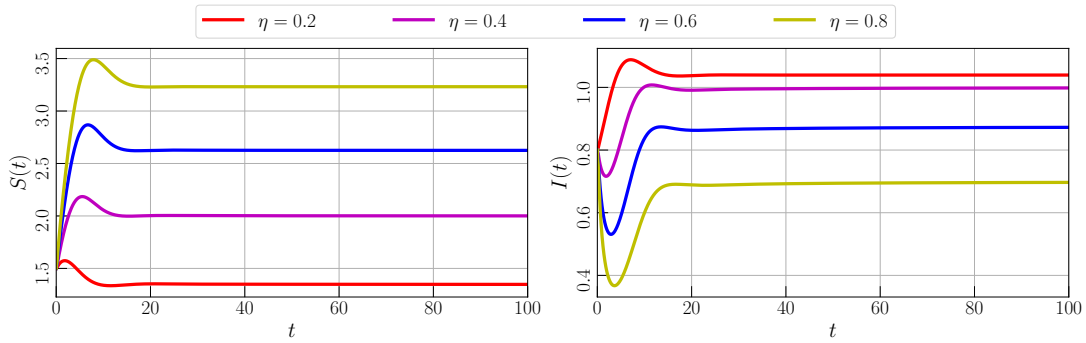
(A) Bifurcation diagram driven by η in interval $0 \leq \eta \leq 1$ (B) Time-series for $\eta = 0.2, 0.4, 0.6,$ and 0.8

FIGURE 6. Bifurcation diagram and times-series of model (8) driven by the recovery rate (η) with parameter values given by eq. (20)

bifurcation diagram as in Figure 5a. When $0 \leq \beta < \beta^*$, $\beta^* = 0.16$, the EEP \mathcal{E}_I does not exist and Theorem 7 is satisfied which means that DFE \mathcal{E}_A is LAS. The solution is convergent to \mathcal{E}_A which indicates the population free from disease. When β passes through β^* , \mathcal{E}_A loses its stability, and unique LAS EEP \mathcal{E}_I occurs in the interior. The infectious disease becomes endemic in the population and still exists for all $t \rightarrow \infty$. From the concatenation of those biological circumstances, we conclude that forward bifurcation occurs around \mathcal{E}_A where β is the bifurcation parameter and $\beta = \beta^*$ is the bifurcation point. It is easy to examine that the bifurcation point $\beta = \beta^*$ is equal to $\mathcal{R}_0 = 1$. The dynamical behaviors are maintained for $\beta^* < \beta \leq 1$. To support these conditions, some time series are given in Figure 5b to show the convergence of solutions for different values of β .

Next, the impact of recover rate (η) is studied. A similar numerical scheme as the previous way is applied. To depicts the bifurcation diagram, the parameter is fixed as in eq. (20) and the recovery rate (η) is varied in interval $0 \leq \eta \leq 1$. We have Figure 6a as the result. Denote that the bifurcation does not exist for this interval. Both DFEP and EEP exist with distinct stability. The DFEP \mathcal{E}_A is a saddle point while the EEP \mathcal{E}_I is LAS which confirm the validity of Theorems 6 and 7. We also confirm that the EEP \mathcal{E}_I attains GAS which means that all initial conditions will go right to the EEP and the infectious disease will exist all the time. Although the disease becomes endemic,

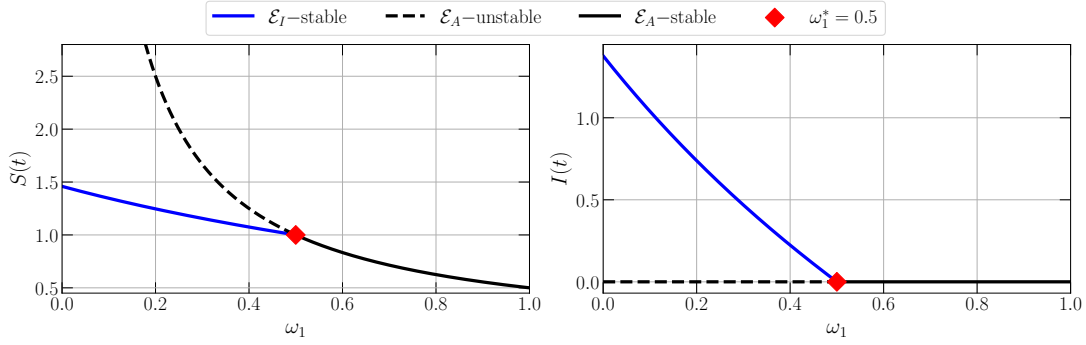
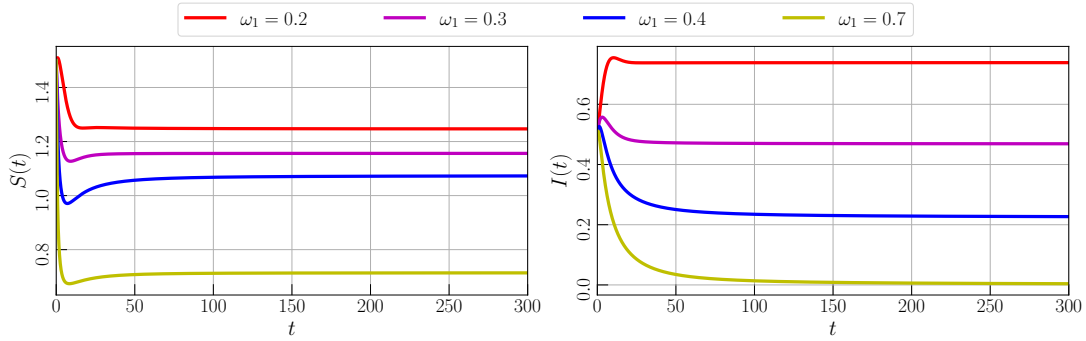

 (A) Bifurcation diagram driven by ω_1 in interval $0 \leq \omega_1 \leq 1$

 (B) Time-series for $\omega_1 = 0.2, 0.3, 0.4$, and 0.7

FIGURE 7. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to intraspecific competition (ω_1) with parameter values given by eq. (20)

the numerical simulation shows that the value of η is directly proportional to $S(t)$ and inversely proportional to $I(t)$, see Figure 6b. This means the population density of the infected class can be reduced by increasing the recovery rate (η).

For the next simulation, the impact of intraspecific competition is investigated. The death rate parameters caused by intraspecific competition on susceptible and infected classes (ω_1 and ω_3) are varied in interval $[0, 1]$. It is found that forward bifurcation occurs when ω_1 is driven where the bifurcation point is given by $\omega_1^* = 0.5$, see Figure 7a. The population density of both susceptible and infected classes reduces when the death rate of $S(t)$ due to intraspecific competition increases as given by Figure 7b. Particularly, Figure 8a shows that bifurcation does not exist in interval $0 \leq \omega_1 \leq 1$ when ω_1 is varied but the dynamical behaviors show that $S(t)$ increases and $I(t)$ decrease when ω_1 increase. We confirm this condition by giving time-series in Figure 8b.

Now, we study the impact of interspecific competition on the dynamical behaviors of model (8). Both susceptible and infected classes have died due to the existence of interspecific competition given by parameters ω_2 and ω_4 . By varying ω_2 and ω_4 in interval $[0, 1]$, we obtain Figures 9a and 10a as the bifurcation diagram. We find

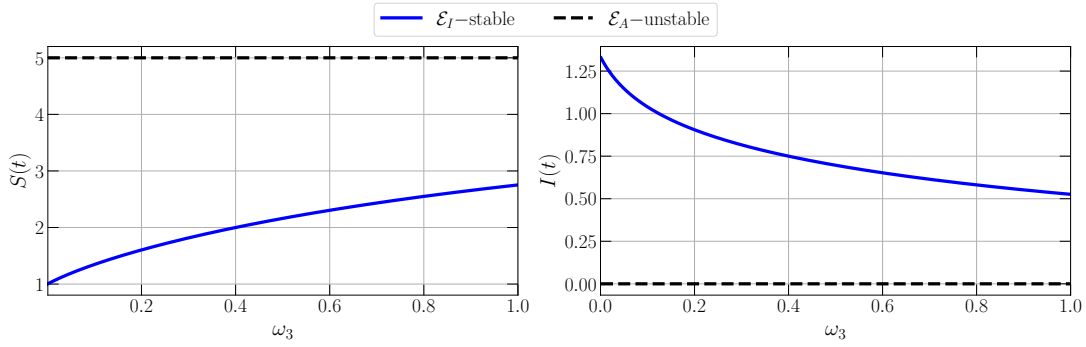
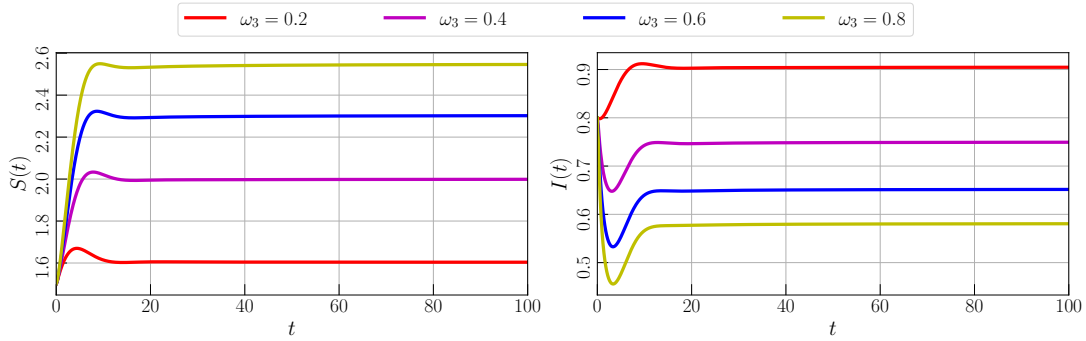
(A) Bifurcation diagram driven by ω_3 in interval $0 \leq \omega_3 \leq 1$ (B) Time-series for $\omega_3 = 0.2, 0.4, 0.6,$ and 0.8

FIGURE 8. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to intraspecific competition (ω_3) with parameter values given by eq. (20)

forward bifurcation driven by ω_4 which does not exist when varying ω_1 . This means, the EEP still exists and LAS for $0 \leq \omega_2 \leq 1$. The EEP will disappear via forward bifurcation and the saddle DFEP becomes LAS when ω_4 crosses $\omega_4^* = 0.34$. This guarantees that the infectious disease may eliminate the disease in population when the death rate of the infected population due to interspecific competition increases as shown in Figure 10b. Although the disease does not disappear when ω_2 is driven, we also can see in Figure 9b that by increasing ω_2 , the population density of the infected class will reduce and the susceptible class will increase.

Finally, the impact of memory effect (α) is investigated. The numerical simulation is given by Figure 11. For $\alpha = 0.7, 0.8, 0.9, 1$ and similar initial values, all solution converge to single equilibrium point given by $\mathcal{E}_I \approx (1.3465, 1.0395)$, see Figure 11(a,b). We then plot the local amplification to show the difference of solutions when α is varied. We find that the difference lies in the convergence rate where for larger values of α , the convergence rate increase and vice versa as shown in Figure 11(e,f). In the beginning, Figure 11(c,d) we show that when α decrease, the population density of the infected class reduce. From a biological point of view, we can say that biological memory has an impact on the density of both susceptible and infected classes.

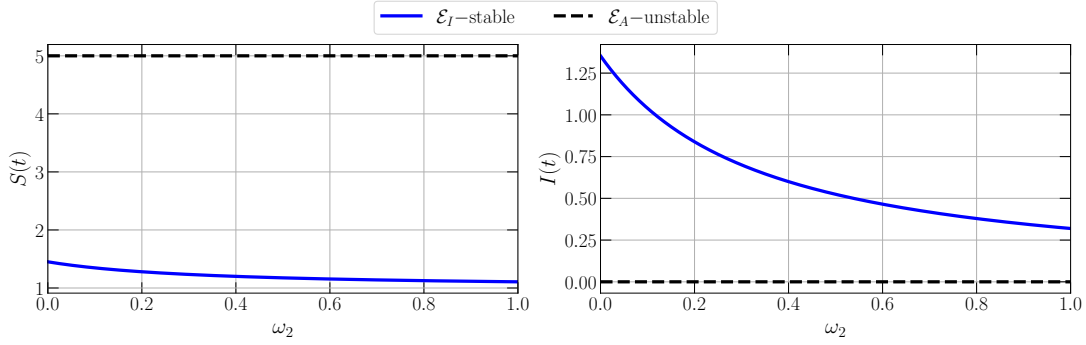
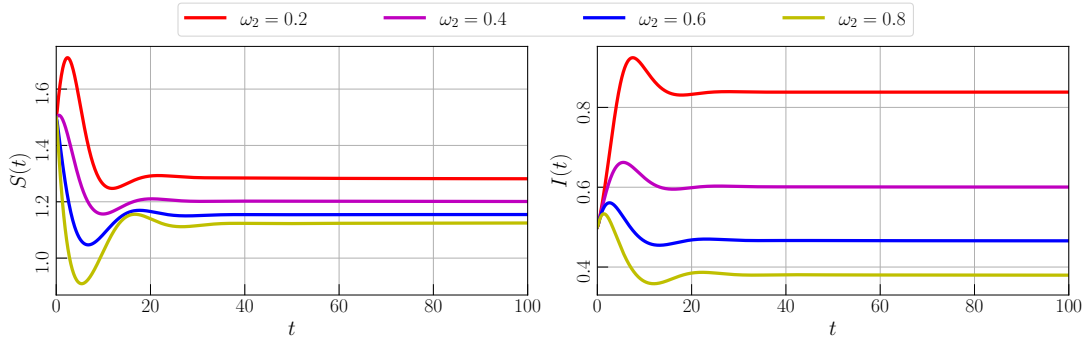
(A) Bifurcation diagram driven by ω_2 in interval $0 \leq \omega_2 \leq 1$ (B) Time-series for $\omega_2 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 9. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to interspecific competition (ω_2) with parameter values given by eq. (20)

6. CONCLUSION

The dynamics of a fractional-order SIS-epidemic model with intraspecific and interspecific competition have been studied. The validity of the model has been confirmed analytically by showing the existence, uniqueness, non-negativity, and boundedness of solutions. Three equilibrium points have been obtained namely the origin, the disease-free equilibrium point, and the endemic equilibrium point. Both origin and disease-free equilibrium points always exist while the endemic equilibrium point conditionally exists. The basic reproduction number \mathcal{R}_0 has been given which has a relationship with the local stability of the model. If $\mathcal{R}_0 < 1$ then the disease-free equilibrium point is locally asymptotically stable and if $\mathcal{R}_0 > 1$ then the disease-free equilibrium point loses its stability along with the existence of a locally asymptotically stable endemic equilibrium point. The global stability conditions of equilibrium points also have been found. The PRCC has been worked to investigate the most influential parameter. We have successfully shown that the infection rate and the death rate of the infected population due to interspecific competition becomes the most influential parameter for basic reproduction number and the population density of

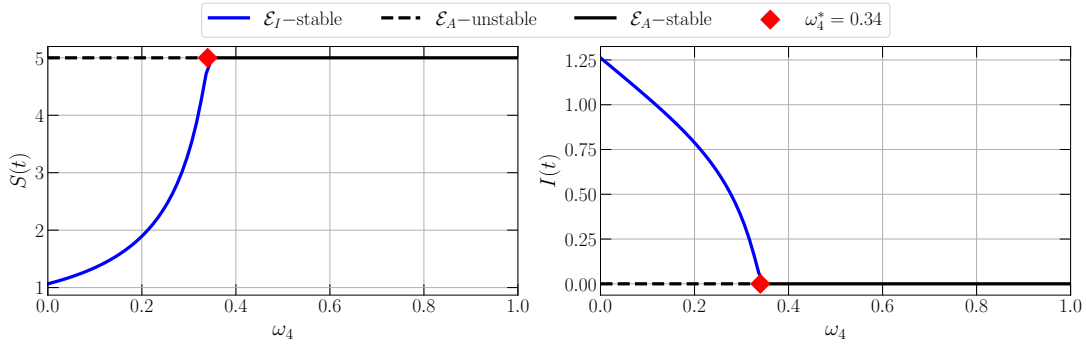
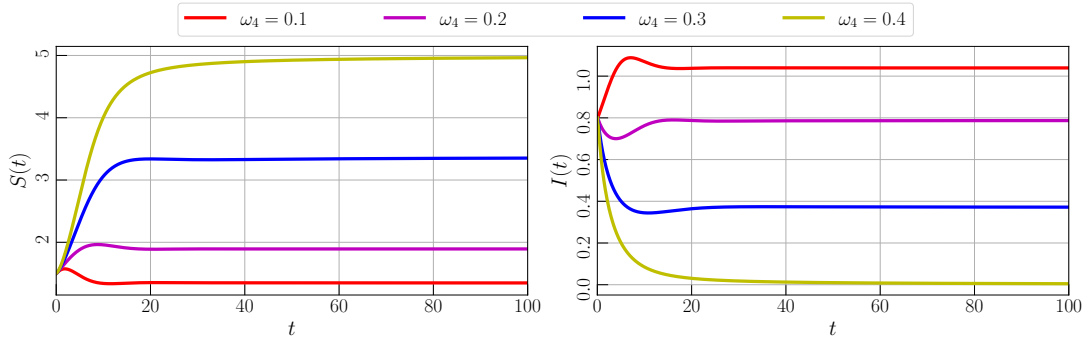
(A) Bifurcation diagram driven by ω_4 in interval $0 \leq \omega_4 \leq 1$ (B) Time-series for $\omega_4 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 10. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to interspecific competition (ω_4) with parameter values given by eq. (20)

the infected class. We then investigate the impact of several parameters using numerical simulations including the infection rate, the recovery rate, the intraspecific competition, the interspecific competition, and the memory effect on the dynamics of the model. Bifurcation diagrams and time series have been given which show the existence of forward bifurcation, the decrease of susceptible and infected classes, and the decrease of convergence rate caused by the memory effect.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

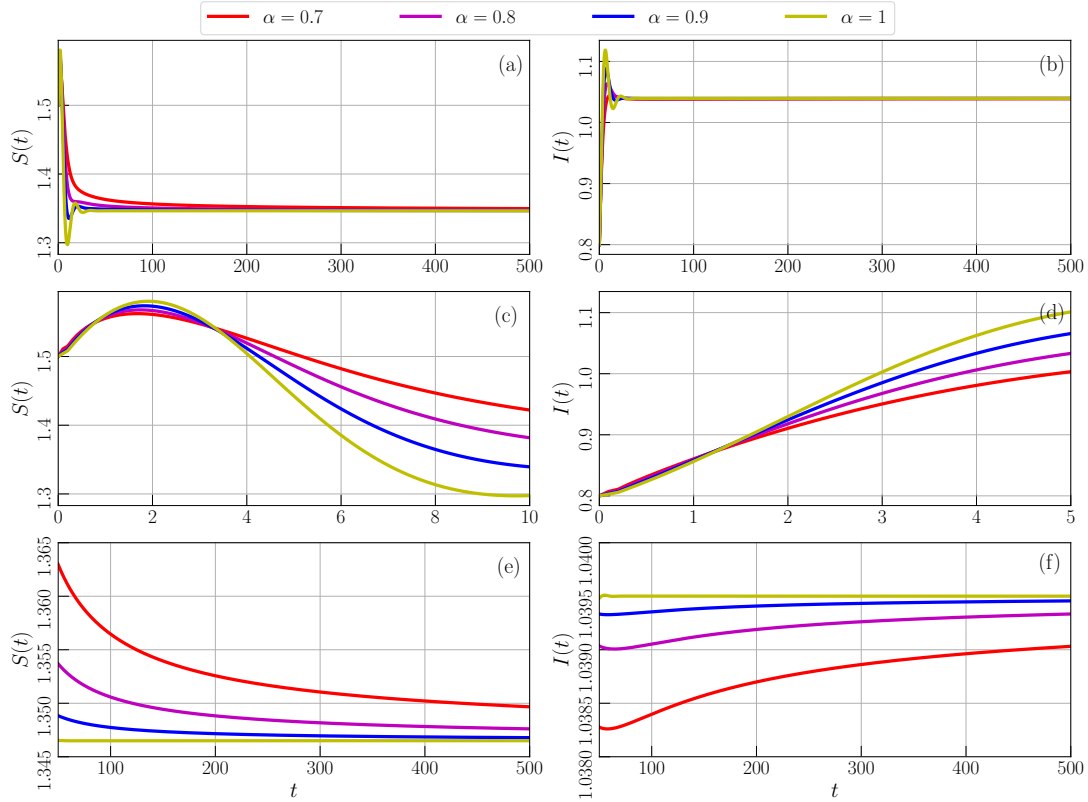


FIGURE 11. Time series of model (8) with parameter values given by eq. (20) for $\alpha = 0.7, 0.8, 0.9, 1$. **(a,b)** Time-series for $0 \leq t \leq 500$, **(c,d)** Local amplification of (a,b) around $0 \leq t \leq 10$, and **(e,f)** Local amplification of (a,b) around $100 \leq t \leq 500$

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DYNAMICS OF SIS–EPIDEMIC MODEL WITH COMPETITION INVOLVING FRACTIONAL-ORDER DERIVATIVE WITH POWER-LAW KERNEL

ISMAIL DJAKARIA^{1,*}, HASAN S. PANIGORO², EBENEZER BONYAH³, EMLI RAHMI², WAHAB MUSA⁴

¹Magister Mathematics Education Programme, Post-Graduate, Universitas Negeri Gorontalo,
Gorontalo 96128, Indonesia

²Biomathematics Research Group, Department of Mathematics, Faculty of Mathematics and Natural Sciences,
Universitas Negeri Gorontalo, Bone Bolango 96119, Indonesia

³Department of Mathematics Education, Akenten Appiah-Menka University of Skills Training and
Entrepreneurial Development, Kumasi 00233, Ghana

⁴Department of Electrical Engineering, Universitas Negeri Gorontalo, Gorontalo 96128, Indonesia

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Abstract. Infectious disease and competition play important roles in the dynamics of a population due to their capability to increase the mortality rate for each organism. In this paper, the dynamical behaviors of a single species population are studied by considering the existence of the infectious disease, intraspecific competition, and interspecific competition. The fractional-order derivative with a power-law kernel is utilized to involve the impact of the memory effect. The population is divided into two compartments namely the susceptible class and the infected class. The existence, uniqueness, non-negativity, and boundedness of the solution are investigated to confirm the biological validity. Three types of feasible equilibrium points are identified namely the origin, the disease-free, and the endemic points. All biological conditions which present the local and global stability are investigated. The global sensitivity analysis is given to investigate the most influential parameter to the basic reproduction number and the density of each class. Some numerical simulations including bifurcation diagrams and time series are also portrayed to explore more the dynamical behaviors.

*Corresponding author

E-mail address: iskar@ung.ac.id

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24 **Keywords:** infectious disease; competition; fractional derivative; Caputo operator; dynamical behaviors.

25 **2010 AMS Subject Classification:** 34A34, 92D30, 37N25, 37N30, 92B05.

26 **1. INTRODUCTION**

27 The spread of infectious disease still becomes a fundamental issue not only because of the
28 existence of the population but also to maintain the balance of biological systems. Several sci-
29 entific methods are developed to discover better ways to suppress and control the rate of disease
30 infection [1]. The preferred ways for the last decades for this epidemiological problems are
31 given by mathematical approach using a deterministic model which is considered efficacious to
32 understand the mechanisms of disease transmission and evaluate the appropriate control strate-
33 gies [2, 3, 4]. The fundamental one which has become the basis of epidemiological modeling
34 is given by [5] which develops the continuous-time deterministic model using first-order de-
35 rivative as the operator. This model is successfully developed in couple of ways such as the
36 continuous-time single species epidemiological modeling with first-order derivative [6, 7, 8, 9],
37 the discrete-time single species epidemiological modeling [10, 11, 12], the stochastic single-
38 species epidemiological modeling [13, 14], and the continuous-time eco-epidemiological mod-
39 eling [15, 16, 17].

40 Apart from those operators, several researchers prefer to use the fractional-order derivative
41 to accomplish their problems the biological modeling. See [18, 19, 20] and references therein
42 for some examples in epidemiological modeling. The fractional-order derivative is chosen by
43 considering the capability of this operator to describe the current state of the biological object
44 as the impact of all of its previous conditions which are known as the memory effect [21, 22]. In
45 the epidemiological model, the transmission of disease may slow down and be forestalled by the
46 susceptible population as the impact of the memory [23]. Some fractional-order derivative has
47 been developed and successfully applied in epidemiological modeling such as the Riemann-
48 Liouville, Caputo, Caputo-Fabrizio, and Atangana-Baleanu [24, 25, 26, 27]. From all of the
49 given operators, the Caputo fractional-order derivative has the complete tools for dynamical
50 analysis such as the existence and uniqueness, non-negativity and boundedness, local dynamics,
51 global dynamics, and some bifurcation analysis. Consequently, the Caputo operator will be used
52 in this paper where defined later in the next section.

53 In this work, we develop the epidemiological model based on the SIR model given by [5]. For
54 single-species conditions, this model is only popular for the infectious diseases that appeared
55 in the human population. In facts, infectious diseases also threaten the existence of the animal
56 population which disturbs the balance of the ecosystem. For examples, the infectious diseases
57 in endemic species such as Orangutans [28], Tarsius [29], Sumatran Tiger [30], and Komodo
58 dragon [31]. Moreover, the natural behaviors of animals that endanger the existence of their
59 populations are the intraspecific competition among them to preserve their food sources [32,
60 33, 34]. For these reasons, developing and investigating the dynamics of the epidemiological
61 model by considering the impact of intraspecific competition and the memory effect are critical
62 issues that become the novelty of our research.

63 The whole of this paper is organized in the following procedure: In Section 2, the math-
64 ematical modeling consists of model formulation, existence, uniqueness, non-negativity, and
65 boundedness are given. The analytical results including the existence of equilibrium points and
66 their local and global dynamics are completely investigated in Section 3. To show the most in-
67 fluent parameter of the model, the global sensitivity analysis is provided by Section 4. Some
68 numerical simulations as well as bifurcation diagrams and time-series are presented in Sec-
69 tion 5 to explore more about the dynamical behaviors of the model. This work ends by giving a
70 conclusion in Section 6.

71 **2. MATHEMATICAL MODELING**

72 This section studies about mathematical modeling consisting of the model formulation, ex-
73 istence, uniqueness, non-negativity, and boundedness of solution. The mathematical model is
74 constructed by a deterministic approach using a differential equation. We first give some as-
75 sumptions to restrain the model so it does not get too complicated. We next interpret the giving
76 assumptions to the mathematical formula using the first-order derivative as the operator. A di-
77 agram is presented to show the impact of each assumption on the flow of population density
78 for each compartment. To involve the impact of the memory effect, the Caputo fractional-order
79 derivative is applied to the model. For the mathematical model's validity, we show that the
80 solution of the model always exists, unique, non-negative, and bounded.

81 **2.1. Model Formulation.** In this work, the model is constructed from a single population
 82 growth model. We first assume there exists a population in a habitat that grows proportionally
 83 to its density and bounded due to the intraspecific competition. Let $N(t)$ be the population
 84 at time t , r is the birth rate, μ is the natural death rate, and ω is the death rate as a result of
 85 competition. Thus, we have a first-order differential equation as follows.

$$86 \quad (1) \quad \frac{dN}{dt} = (r - \mu)N - \omega N^2.$$

87 Next, we assume that the population is exposed by infectious disease. The population N is
 88 divided into two compartments namely the susceptible class (S) and infected class (I) where
 89 $N = S + I$. The susceptible class is infected by disease bilinearly with infection rate β . The
 90 competition is divided into two cases namely the intraspecific competition for each susceptible
 91 and infected class, and the interspecific competition between susceptible and infected classes.
 92 As result, the following model is received.

$$93 \quad (2) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - \mu I, \end{aligned}$$

94 where ω_i , $i = 1, 2$ respectively denote the death rate of the susceptible population as the results
 95 of intraspecific and interspecific competitions between susceptible and susceptible classes, and
 96 susceptible and infected classes. The parameters ω_i , $i = 3, 4$ denote the death rate of the infected
 97 population as the result of competition between infected and infected classes, and susceptible
 98 and infected classes. In our works, we also assume that each organism has the capability to
 99 survive the disease. Thus, we define η as the recovery rate. Since each organism that survives
 100 from the disease has a chance to be re-infected, this type of population will be again susceptible.
 101 Finally, we have a mathematical model as follows.

$$102 \quad (3) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

103 All of the given assumptions and their mathematical modeling are described in Figure 1.

104 Now, the Caputo fractional-order derivative will be applied in order to conduct the impact
 105 of the memory effect on the population growth rate. The similar procedure is adopted from

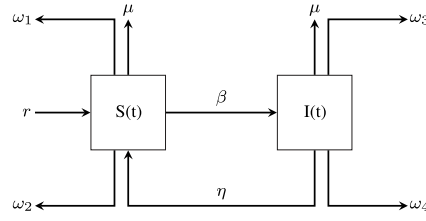


FIGURE 1. Compartment diagram of model (3)

106 [35]. The first-order derivatives on the left-hand side of model (3) are replaced by the Caputo
 107 fractional-order derivative defined as follows.

108 **Definition 1.** [36] Suppose $0 < \alpha \leq 1$. The Caputo fractional derivative of order $-\alpha$ is defined
 109 by

$$110 \quad (4) \quad {}^C \mathcal{D}_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} f'(s) ds,$$

111 where $t \geq 0$, $f \in C^n([0, +\infty), \mathbb{R})$, and Γ is the Gamma function.

112 Applying Definition 1 to eq. (3), the following model is obtained.

$$113 \quad (5) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

114 Since the given process above makes the dimension of time at the left-hand side become t^α ,
 115 some parameters need to be rescaled so that there are no differences between the time's dimen-
 116 sions at the left-hand side with the right-hand side of model (5). By applying time rescale to
 117 some parameters, we have the model as follows.

$$118 \quad (6) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r^\alpha - \mu^\alpha)S - \omega_1^\alpha S^2 - (\omega_2^\alpha + \beta^\alpha)SI + \eta^\alpha I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta^\alpha - \omega_4^\alpha)SI - \omega_3^\alpha I^2 - (\eta^\alpha + \mu^\alpha)I. \end{aligned}$$

119 Let $r^\alpha = \hat{r}$, $\mu^\alpha = \hat{\mu}$, $\omega_1^\alpha = \hat{\omega}_1$, $\omega_2^\alpha = \hat{\omega}_2$, $\omega_3^\alpha = \hat{\omega}_3$, $\omega_4^\alpha = \hat{\omega}_4$, $\beta^\alpha = \hat{\beta}$, and $\eta^\alpha = \hat{\eta}$. Thus, we
 120 acquire

$$121 \quad (7) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (\hat{r} - \hat{\mu})S - \hat{\omega}_1 S^2 - (\hat{\omega}_2 + \hat{\beta})SI + \hat{\eta}I, \\ {}^C \mathcal{D}_t^\alpha I &= (\hat{\beta} - \hat{\omega}_4)SI - \hat{\omega}_3 I^2 - (\hat{\eta} + \hat{\mu})I. \end{aligned}$$

122 For simplicity, by dropping $\hat{\cdot}$ for each parameter, we obtain the final model as follows.

$$\begin{aligned}
 123 \quad (8) \quad {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I = F_1(N(t)), \\
 {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I = F_2(N(t)).
 \end{aligned}$$

124 Equation (8) is the final proposed model in this paper. Although model (8) seems classic and
 125 simple, this model will be powerful to solve and investigate the existence of a closed population
 126 in a certain area without any outside intervention. Our literature review also shows that the
 127 model (8) has heretofore never been studied. Now, the basic properties of model (8) such as the
 128 existence uniqueness, non-negativity, and boundedness are investigated to confirm its biological
 129 validity.

130 **2.2. Existence and Uniqueness.** In this subsection, we will show that the model (8) has a
 131 unique solution. A similar manner given by [37] is used. Thus, the following theorem is
 132 presented to show the existence and uniqueness of the solution of model (8).

133 **Theorem 1.** *The model (8) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ has a unique*
 134 *solution.*

Proof. Consider model (8) with positive initial condition with $F : [0, \infty) \rightarrow \mathbb{R}^2$ where $F(N) = (F_1(N), F_2(N))$, $N \equiv N(t)$ and $\theta \equiv \{(S, I) \in \mathbb{R}_+^2 : \max\{|S|, |I|\} \leq M\}$ for sufficiently large M . Then, for any $N = (S, I)$ and $\bar{N} = (\bar{S}, \bar{I})$, $N, \bar{N} \in \theta$, we have

$$\begin{aligned}
 & \|F(N) - F(\bar{N})\| \\
 &= |F_1(N) - F_1(\bar{N})| + |F_2(N) - F_2(\bar{N})| \\
 &= \left| [(r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I] - [(r - \mu)\bar{S} - \omega_1 \bar{S}^2 - (\omega_2 + \beta)\bar{S}\bar{I} + \eta \bar{I}] \right| + \\
 & \quad \left| [(\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I] - [(\beta - \omega_4)\bar{S}\bar{I} - \omega_3 \bar{I}^2 - (\eta + \mu)\bar{I}] \right| \\
 &\leq (r + \mu)|S - \bar{S}| + \omega_1 |S^2 - \bar{S}^2| + (\omega_2 + \beta)|SI - \bar{S}\bar{I}| + \eta |I - \bar{I}| + (\beta + \omega_4)|SI - \bar{S}\bar{I}| \\
 & \quad + \omega_3 |I^2 - \bar{I}^2| + (\eta + \mu)|I - \bar{I}| \\
 &= (r + \mu)|S - \bar{S}| + \omega_1 |(S + \bar{S})(S - \bar{S})| + (\omega_2 + \omega_4 + 2\beta)|I(S - \bar{S}) + \bar{S}(I - \bar{I})| \\
 & \quad + (2\eta + \mu)|I - \bar{I}| + \omega_3 |(I + \bar{I})(I - \bar{I})|
 \end{aligned}$$

$$\begin{aligned}
&\leq (r + \mu) |S - \bar{S}| + 2\omega_1 M |S - \bar{S}| + (\omega_2 + \omega_4 + 2\beta) M |S - \bar{S}| \\
&\quad + (\omega_2 + \omega_4 + 2\beta) M |I - \bar{I}| + (2\eta + \mu) |I - \bar{I}| + 2\omega_3 M |I - \bar{I}| \\
&= [(r + \mu) + 2\omega_1 M + (\omega_2 + \omega_4 + 2\beta) M] |S - \bar{S}| \\
&\quad + [(\omega_2 + \omega_4 + 2\beta) M + (2\eta + \mu) + 2\omega_3 M] |I - \bar{I}| \\
&\leq L \|N - \bar{N}\|,
\end{aligned}$$

135 where $L = (\omega_2 + \omega_4 + 2\beta) M + \mu + \max\{r + 2\omega_1 M, 2(\eta + \omega_3 M)\}$. Therefore, $F(N)$ satisfies the
136 Lipschitz condition. Obeying Lemma 5 in [38], we conclude that model (8) with positive initial
137 condition has a unique solution. \square

138 **2.3. Non-negativity and Boundedness.** The non-negativity and boundedness properties of
139 the solutions of the model (8) are given in the following theorem.

140 **Theorem 2.** *All solution of the model (8), which start in $\mathbb{R}_+^2 :=$
141 $\{(S, I) \mid S \geq 0, I \geq 0, (S, I) \in \mathbb{R}^2\}$ are uniformly bounded and non-negative.*

Proof. To prove the boundedness of the solutions of the model (8), the same approach of [38]
is adopted. Let consider the function $N = S + I$. Then,

$$\begin{aligned}
{}^C \mathcal{D}_t^\alpha N &= {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I + (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I.
\end{aligned}$$

Hence, for each $\mu > 0$,

$$\begin{aligned}
{}^C \mathcal{D}_t^\alpha N + \mu N &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I + \mu S + \mu I \\
&= rS - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&= -\omega_1 \left(S - \frac{r}{2\omega_1} \right)^2 + \frac{r^2}{4\omega_1} - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&\leq \frac{r^2}{4\omega_1}
\end{aligned}$$

142 By using the comparison theorem in [39], we obtain $N(t) \leq N(0)E_\alpha(-\mu t^\alpha) +$
 143 $\frac{r^2}{4\omega_1}t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha)$, where E_α and $E_{\alpha,\alpha+1}$ is the Mittag-Leffler function with one and two
 144 parameters. According to Lemma 5 and Corollary 6 in [39], we have $N(t) \leq \frac{r^2}{4\mu\omega_1}$, as $t \rightarrow \infty$.
 145 Therefore, all solutions of model (8) starting in \mathbb{R}_+^2 are uniformly bounded in the region Φ ,
 146 where $\Phi = \left\{ (S, I) \in \mathbb{R}_+^2 : S + I \leq \frac{r^2}{4\mu\omega_1} + \varepsilon, \varepsilon > 0 \right\}$ Next, we prove that all solutions of model
 147 (8) are non-negative. By model (8), we have ${}^C \mathcal{D}_t^\alpha S|_{S=0} = \eta I \geq 0$ and ${}^C \mathcal{D}_t^\alpha I|_{I=0} = 0 \geq 0$. Based
 148 on Lemmas 5 and 6 in [40], we conclude that the solutions of model (8) are non-negative. \square

149 3. ANALYTICAL RESULTS

150 In this section, the dynamics of model (8) are shown analytically including the existence of
 151 equilibrium points, and their local and global stability.

3.1. Existence of Equilibrium Points. To find the equilibrium points of model (8), we must
 have

$$(9) \quad [(r - \mu) - \omega_1 S - (\omega_2 + \beta)I]S + \eta I = 0,$$

$$(10) \quad [(\beta - \omega_4)S - \omega_3 I - (\eta + \mu)]I = 0.$$

152 If $I = 0$ is substituted to (9), we obtain

$$153 (11) \quad [(r - \mu) - \omega_1 S]S = 0.$$

154 From eq. (11), we get $S = 0$ and $S = \frac{r-\mu}{\omega_1}$. Thus, we have two equilibrium points here namely
 155 $\mathcal{E}_0 = (0, 0)$, and $\mathcal{E}_A = \left(\frac{r-\mu}{\omega_1}, 0 \right)$. The equilibrium point \mathcal{E}_0 is called the origin point which
 156 represents the extinction of both susceptible and infected populations. Since $\mathcal{E}_0 \in \mathbb{R}_+^2$, this
 157 equilibrium point always exists. Furthermore, the equilibrium point \mathcal{E}_A is called the disease-
 158 free equilibrium point (DFEP) which describes the condition where the infectious disease does
 159 not exist anymore in the population. According to the biological condition, it is natural that the
 160 birth rate r is greater than its death rate μ . By assuming $r > \mu$, the origin point $\mathcal{E}_A \in \mathbb{R}_+^2$ also
 161 always exists. By simple calculation, we also obtain the basic reproduction number \mathcal{R}_0 given
 162 by

$$163 (12) \quad \mathcal{R}_0 = \frac{(r - \mu)\beta}{(r - \mu)\omega_4 + (\eta + \mu)\omega_1}.$$

164 The basic reproduction number is utilized to show the dynamical behavior of each equilibrium
 165 point and to describe whether the infectious disease becomes endemic or not. Since $r > \mu$, the
 166 value of \mathcal{R}_0 is always positive. Now, let's concern the eq. (9) and (10). By solving eq. (10), we
 167 attain

$$168 \quad (13) \quad S = \frac{\omega_3 I + (\eta + \mu)}{\beta - \omega_4}.$$

169 If we substitute eq. (13) to (9), the following polynomial equation holds.

$$170 \quad (14) \quad k_1 I^2 + k_2 I + k_3 = 0,$$

where

$$\begin{aligned} k_1 &= ((\beta - \omega_4)(\beta + \omega_2) + \omega_1 \omega_3) \omega_3, \\ k_2 &= (\beta - \omega_4)((\beta + \omega_2)\mu + (\omega_2 + \omega_4)\eta - (r - \mu)\omega_3) + 2(\eta + \mu)\omega_1 \omega_3, \\ k_3 &= \frac{(1 - \mathcal{R}_0)(r - \mu)(\eta + \mu)\beta}{\mathcal{R}_0}. \end{aligned}$$

171 Therefore, we acquire the endemic point (EEP)

$$172 \quad (15) \quad \mathcal{E}_I = \left(\frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}, \bar{\gamma} \right),$$

173 where $\bar{\gamma}$ is the positive root of polynomial equation (14). From (15), we find that $\beta > \omega_4$ must
 174 be fulfilled so that $\mathcal{E}_I \in \mathbb{R}_+^2$. Moreover, EEP exists if $\bar{\gamma} > 0$. From eq. (14), we have k_1 is always
 175 positive. Thus, the value of the $\bar{\gamma}$ depends on k_2 and k_3 . Furthermore, eq. (14) has real number
 176 roots if $k_2^2 \geq 4k_1 k_3$. By applying simple algebra, if $k_3 > 0$ and $k_2 < 0$ then we have two positive
 177 roots of eq. (14), if $k_3 > 0$ and $k_2 > 0$ then we do not have any positive roots of eq. (14), and if
 178 $k_3 < 0$ then we have a positive root of eq. (14). Finally, we have the following theorem.

179 **Theorem 3.** *Let $\beta > \omega_4$. The existence of EEP \mathcal{E}_I is shown by the following statement.*

180 (i) *If $k_2^2 < 4k_1 k_3$ then \mathcal{E}_I does not exist.*

181 (ii) *If $k_2^2 = 4k_1 k_3$ and*

182 (ii.i) *if $k_2 > 0$ then \mathcal{E}_I does not exist.*

183 (ii.ii) *if $k_2 < 0$ then \mathcal{E}_I exists and unique.*

184 (iii) *If $k_2^2 > 4k_1 k_3$ and*

185 (iii.i) if $k_3 > 0$ and $k_2 < 0$ then we have a pair of \mathcal{E}_1 .

186 (iii.ii) if $k_3 > 0$ and $k_2 > 0$ then \mathcal{E}_1 does not exist.

187 (iii.iii) if $k_3 < 0$ then \mathcal{E}_1 exists and unique.

188 Denote that $k_2^2 > 4k_1k_3$ is always satisfied and $k_3 < 0$ for $\mathcal{R}_0 > 1$, then the following lemma
189 holds.

190 **Lemma 4.** *EEP \mathcal{E}_1 exists and unique if $\mathcal{R}_0 > 1$.*

191 **3.2. Local Dynamics.** The local dynamics of model (8) are obtained by applying the
192 Matignon condition which is defined as follows.

193 **Theorem 5.** [Matignon condition [36]] *An equilibrium point \vec{x}^* is locally asymptotically stable*
194 *(LAS) if all eigenvalues λ_j of the Jacobian matrix $J = \frac{\partial \vec{f}}{\partial \vec{x}}$ at \vec{x}^* satisfy $|\arg(\lambda_j)| > \frac{\alpha\pi}{2}$. If there*
195 *exists at least one eigenvalue satisfy $|\arg(\lambda_k)| > \frac{\alpha\pi}{2}$ while $|\arg(\lambda_l)| < \frac{\alpha\pi}{2}$, $k \neq l$, then \vec{x}^* is a*
196 *saddle-point.*

197 Therefore, to study the local dynamics of model (8), we first compute its Jacobian matrix at
198 the point (S, I) which gives

$$199 \quad (16) \quad \mathcal{J}(S, I) = \begin{bmatrix} (r - \mu) - 2\omega_1 S - (\omega_2 + \beta)I & -(\omega_2 + \beta)S + \eta \\ (\beta - \omega_4)I & (\beta - \omega_4)S - 2\omega_3 I - (\eta + \mu) \end{bmatrix}.$$

200 Obeying Theorem 5 and using Jacobian matrix (16), we discuss the local stability for each
201 equilibrium point in the next subsection.

3.3. Dynamical behavior around \mathcal{E}_0 . LAS condition of \mathcal{E}_0 is obtained by identifying the
eigenvalues of the Jacobian matrix (16) at the point $(S, I) = (0, 0)$. We receive

$$\mathcal{J}(S, I)|_{\mathcal{E}_0} = \begin{bmatrix} r - \mu & \eta \\ 0 & -(\eta + \mu) \end{bmatrix}.$$

202 Therefore, we have $\lambda_1 = r - \mu$ and $\lambda_2 = -(\eta + \mu)$. Since $r > \mu$ and $\lambda_2 < 0$, we have $|\arg(\lambda_1)| =$
203 $0 < \frac{\alpha\pi}{2}$ and $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$. According to Theorem 5, the following theorem holds.

204 **Theorem 6.** *The origin point \mathcal{E}_0 is always a saddle point.*

3.4. Dynamical behavior around \mathcal{E}_A . For $(x, y) = \left(\frac{r-\mu}{\omega_1}, 0\right)$, the Jacobian matrix (16) becomes

$$\mathcal{J}(S, I)|_{\mathcal{E}_A} = \begin{bmatrix} -(r-\mu) & \eta - \frac{(\omega_2+\beta)(r-\mu)}{\omega_1} \\ 0 & \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0} \end{bmatrix},$$

205 which gives a pair of eigenvalues $\lambda_1 = -(r-\mu)$ and $\lambda_2 = \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0}$. Denote $|\arg(\lambda_2)| =$
 206 $\pi > \frac{\alpha\pi}{2}$ as the impact of $\lambda_1 < 0$. Hence, the sign of λ_2 takes the role in describing local dynamics
 207 around \mathcal{E}_A . To obtain $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$, we need $\lambda_2 < 0$ which is fulfilled if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$
 208 then $|\arg(\lambda_2)| = 0 < \frac{\alpha\pi}{2}$. Following the Matignon condition given in Theorem 5, the following
 209 theorem is successfully attained.

210 **Theorem 7.** *If $\mathcal{R}_0 < 1$ then \mathcal{E}_A is LAS and a saddle point if $\mathcal{R}_0 > 1$.*

211 **3.5. Dynamical behavior around \mathcal{E}_I .** To identify the local stability of \mathcal{E}_I , we first compute
 212 the Jacobian matrix (16) evaluated at \mathcal{E}_I . We generate

$$213 \quad (17) \quad \mathcal{J}(S, I)|_{\mathcal{E}_I} = \begin{bmatrix} -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu}\right] & -\frac{(\omega_2+\beta)(\omega_3\bar{\gamma}+\eta+\mu)}{\beta-\omega_4} + \eta \\ (\beta-\omega_4)\bar{\gamma} & -\omega_3\bar{\gamma} \end{bmatrix}.$$

The eigenvalues of (17) are given by $\lambda_1 = \frac{1}{2} \left(\xi_1 + \sqrt{\xi_1^2 - 4\xi_2} \right)$ and $\lambda_2 = \frac{1}{2} \left(\xi_1 - \sqrt{\xi_1^2 - 4\xi_2} \right)$
 where

$$\begin{aligned} \xi_1 &= -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu} + \omega_3\bar{\gamma} \right], \\ \xi_2 &= \left[\left(\frac{\omega_1\omega_3}{\beta-\omega_4} + \omega_2 + \beta \right) (\omega_3\bar{\gamma} + \eta + \mu) + \left(\frac{\omega_3\bar{\gamma}}{\omega_3\bar{\gamma} + \eta + \mu} + 1 \right) (\beta - \omega_4)\eta \right] \bar{\gamma}. \end{aligned}$$

214 It is easy to proof that $\xi_1 < 0$ and $\xi_2 > 0$ since $\beta > \omega_4$ becomes the existence condition. As the
 215 impact, $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$, $i = 1, 2$ and hence the LAS always hold for EEP. Thus, the following
 216 theorem holds.

217 **Theorem 8.** *EEP \mathcal{E}_I is always LAS.*

218 **3.6. Global Dynamics.** In this subsection, the global dynamics of model (8) are studied. The
 219 biological conditions of equilibrium points are investigated so that those points are globally
 220 asymptotically stable (GAS). Since the origin is always a saddle point, we focus on studying
 221 GAS conditions for DFEP and EEP. The next two theorems are given for the global dynamics.

222 **Theorem 9.** DFEP \mathcal{E}_A is GAS if $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$.

223 *Proof.* We define a positive Lyapunov function as follows.

$$224 \quad (18) \quad \mathcal{V}_A(S, I) = \left(S - \frac{r - \mu}{\omega_1} - \frac{r - \mu}{\omega_1} \ln \frac{\omega_1 S}{r - \mu} \right) + I.$$

If we calculate the Caputo fractional derivative of $\mathcal{V}_A(S, I)$ along the solution of model (8) and use Lemma 3.1 in [41], we get

$$\begin{aligned} & {}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \\ &= \left(\frac{S - \frac{r - \mu}{\omega_1}}{S} \right) {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\ &= -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 + \frac{(r - \mu)(\omega_2 + \beta)I}{\omega_1} - \frac{(r - \mu)\eta I}{\omega_1 S} - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I \\ &\leq -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 - \left(\mu - \frac{(\omega_2 + \beta)r}{\omega_1} \right) I \end{aligned}$$

225 Since $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$, we have ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$, and ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$ only
226 when $(S, I) = \left(\frac{r - \mu}{\omega_1}, 0 \right)$. This means that the singleton $\{\mathcal{E}_A\}$ is the only invariant set where
227 ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$. By Lemma 4.6 in [42], we can conclude that every solution of model (8)
228 tends to DFEP \mathcal{E}_A .

229

□

230 **Theorem 10.** EEP \mathcal{E}_I is GAS if $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$.

231 *Proof.* We first define $\vartheta = \frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}$ and hence $\mathcal{E}_I = (\vartheta, \bar{\gamma})$. Now, a positive Lyapunov function
232 is presented as follows.

$$233 \quad (19) \quad \mathcal{V}_I(S, I) = \left(S - \vartheta - \vartheta \ln \frac{S}{\vartheta} \right) + \left(I - \bar{\gamma} - \bar{\gamma} \ln \frac{S}{\bar{\gamma}} \right)$$

Following Lemma 3.1 in [41], we reach

$$\begin{aligned} & {}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \\ &= \left(\frac{S - \vartheta}{S} \right) {}^C \mathcal{D}_t^\alpha S + \left(\frac{I - \bar{\gamma}}{I} \right) {}^C \mathcal{D}_t^\alpha I \\ &= (S - S^*) \left((r - \mu) - \omega_1 S - (\omega_2 + \beta)I + \frac{\eta I}{S} \right) + (I - \bar{\gamma}) ((\beta - \omega_4)S - \omega_3 I - (\eta + \mu)) \end{aligned}$$

$$\begin{aligned}
 &= -\omega_1 (S - \vartheta)^2 - \omega_3 (I - \bar{\gamma})^2 - (\omega_2 + \omega_4) (S - S^*) (I - \bar{\gamma}) \\
 &\leq -\left(\omega_1 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta}\right)\right) (S - \vartheta)^2 - \left(\omega_3 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta}\right)\right) (I - \bar{\gamma})^2
 \end{aligned}$$

234 Denote that ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$ as a result of $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$. We
 235 also have that ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$ only when $(S, I) = (\vartheta, \bar{\gamma})$. Therefore, the singleton $\{\mathcal{E}_I\}$ is the
 236 only invariant set where ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$. Obeying Lemma 4.6 in [42], every solution of model
 237 (8) tends to EEP \mathcal{E}_I . □

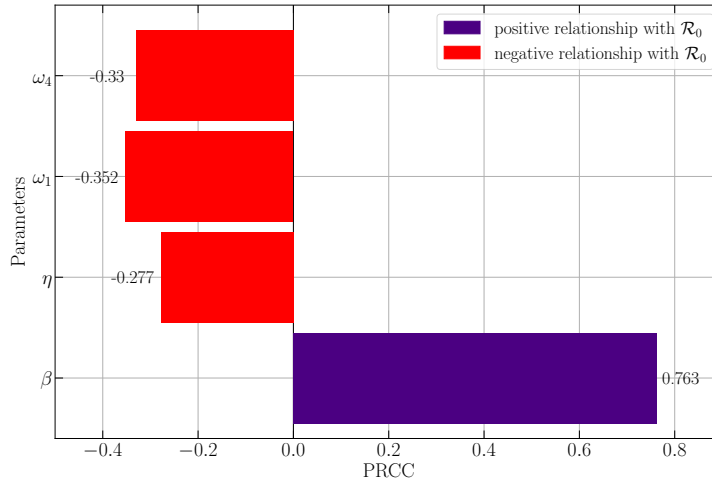


FIGURE 2. PRCC results for the parameters of \mathcal{R}_0

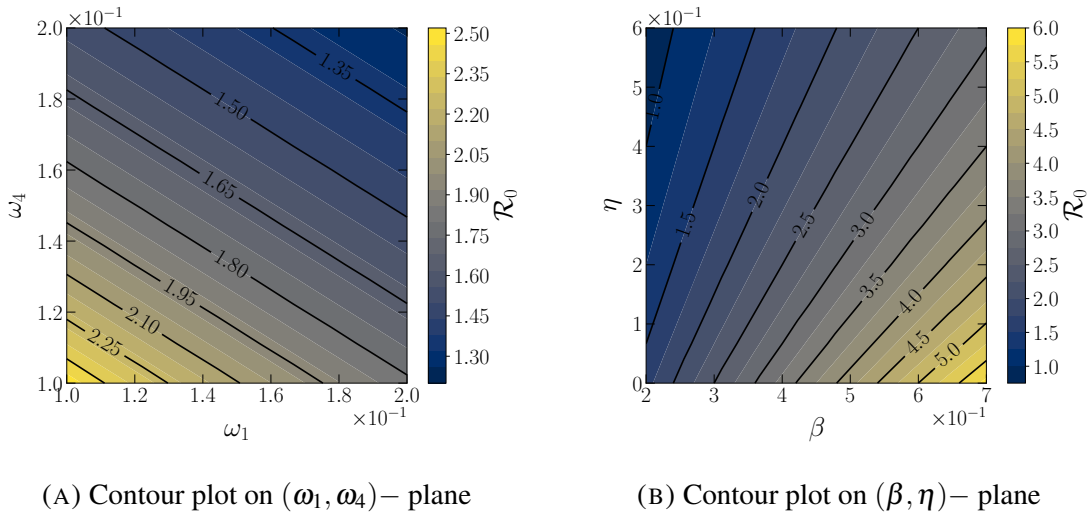


FIGURE 3. Contour plots for the parameters respect to \mathcal{R}_0

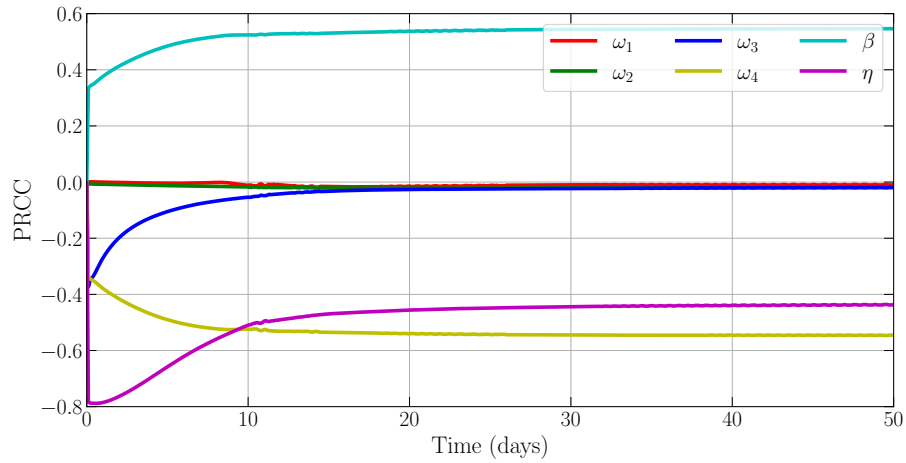
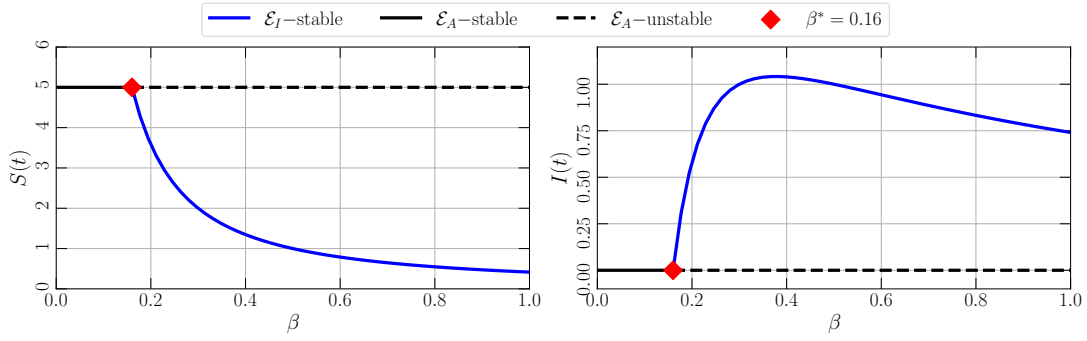
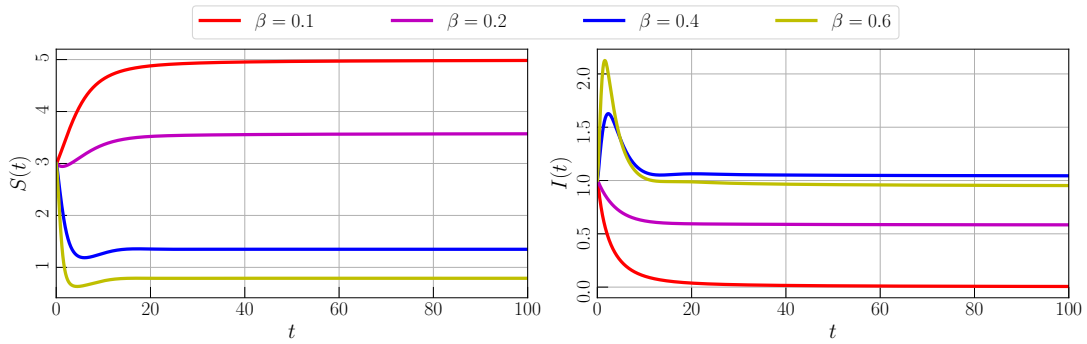
FIGURE 4. PRCC results for the parameters of $I(t)$

TABLE 1. PRCC results in respect to the population density of infected class

Parameter	Description	PRCC	Rank	Relationship with $I(t)$
ω_1	The death rate of susceptible population due to the intraspecific competition	-0.00851	6	Negative relationship
ω_2	The death rate of susceptible population due to the interspecific competition	-0.01938	5	Negative relationship
ω_3	The death rate of infected population due to the intraspecific competition	-0.01990	4	Negative relationship
ω_4	The death rate of infected population due to the interspecific competition	-0.54635	1	Negative relationship
β	The infection rate	0.54631	2	Positive relationship
η	The recovery rate	-0.43606	3	Negative relationship



(A) Bifurcation diagram driven by β in interval $0 \leq \beta \leq 1$



(B) Time-series for $\beta = 0.1, 0.2, 0.4,$ and 0.6

FIGURE 5. Bifurcation diagram and times-series of model (8) driven by the infection rate (β) with parameter values given by eq. (20)

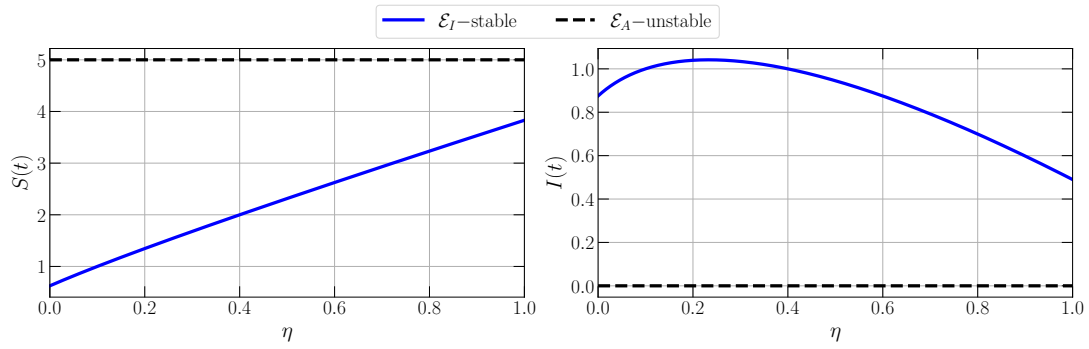
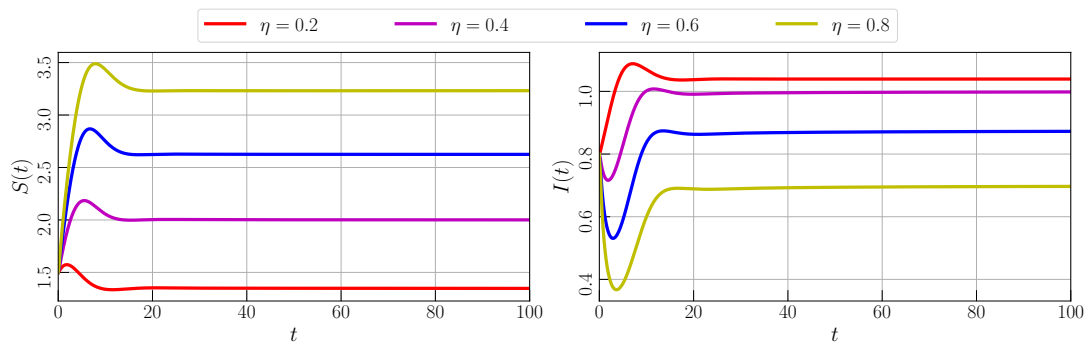
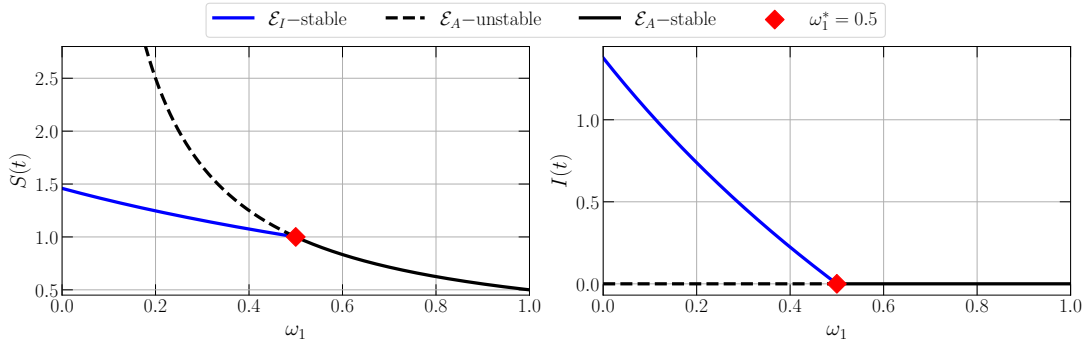
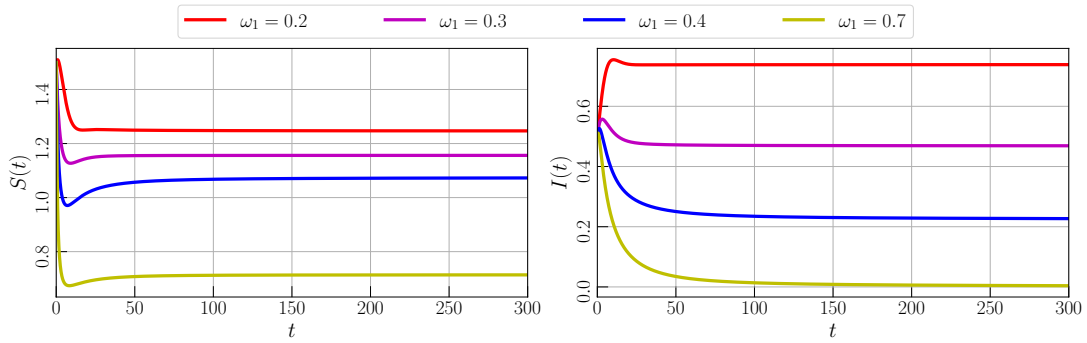
(A) Bifurcation diagram driven by η in interval $0 \leq \eta \leq 1$ (B) Time-series for $\eta = 0.2, 0.4, 0.6,$ and 0.8

FIGURE 6. Bifurcation diagram and times-series of model (8) driven by the recovery rate (η) with parameter values given by eq. (20)



(A) Bifurcation diagram driven by ω_1 in interval $0 \leq \omega_1 \leq 1$



(B) Time-series for $\omega_1 = 0.2, 0.3, 0.4,$ and 0.7

FIGURE 7. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to intraspecific competition (ω_1) with parameter values given by eq. (20)

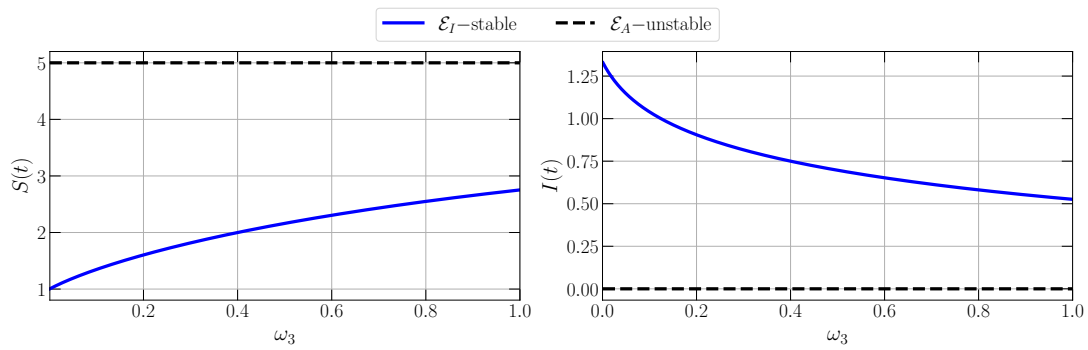
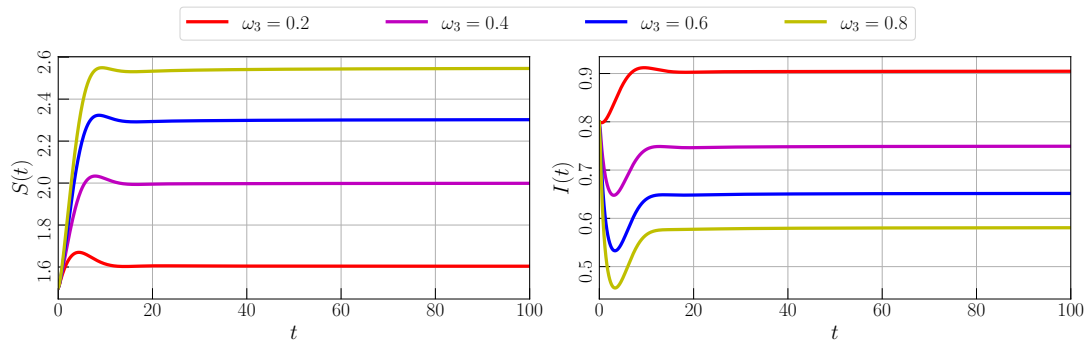
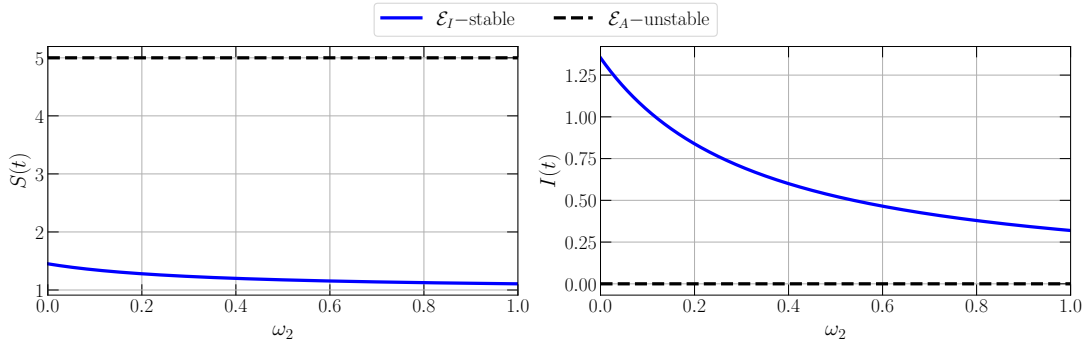
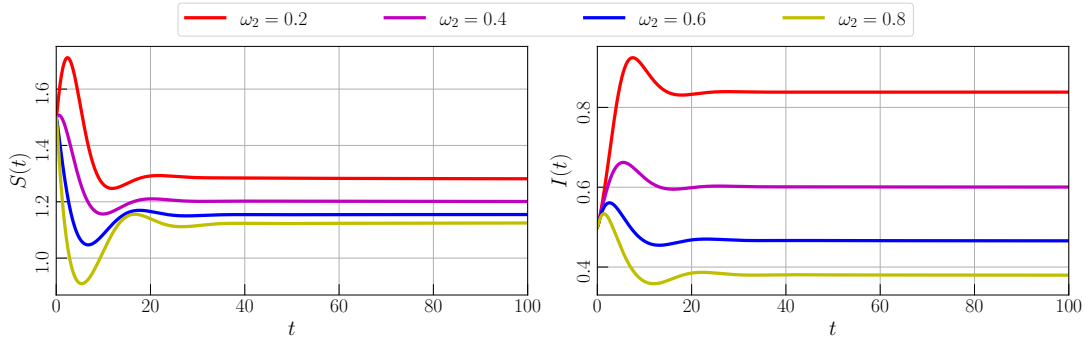
(A) Bifurcation diagram driven by ω_3 in interval $0 \leq \omega_3 \leq 1$ (B) Time-series for $\omega_3 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 8. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to intraspecific competition (ω_3) with parameter values given by eq. (20)



(A) Bifurcation diagram driven by ω_2 in interval $0 \leq \omega_2 \leq 1$



(B) Time-series for $\omega_2 = 0.2, 0.4, 0.6, \text{ and } 0.8$

FIGURE 9. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to interspecific competition (ω_2) with parameter values given by eq. (20)

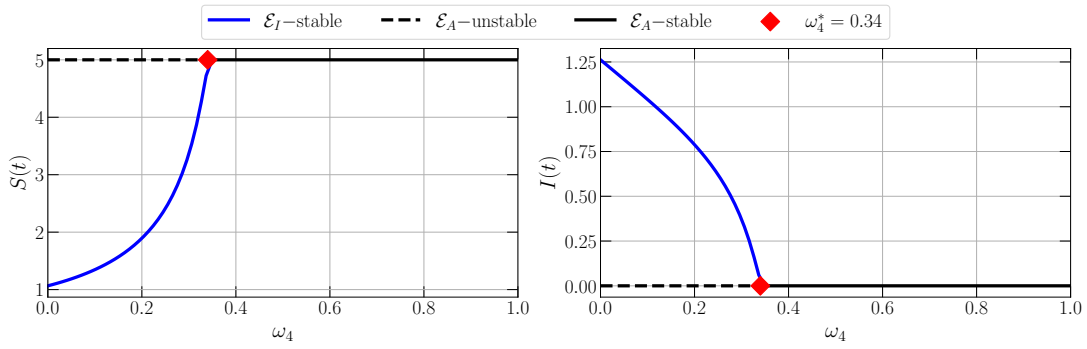
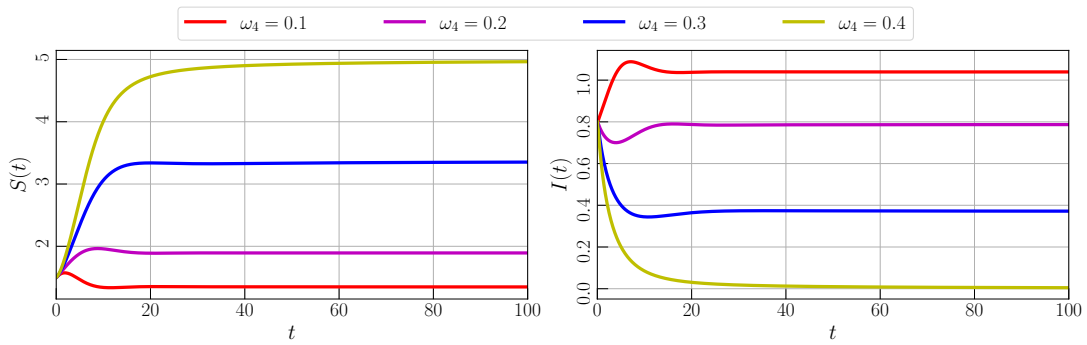
(A) Bifurcation diagram driven by ω_4 in interval $0 \leq \omega_4 \leq 1$ (B) Time-series for $\omega_4 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 10. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to interspecific competition (ω_4) with parameter values given by eq. (20)

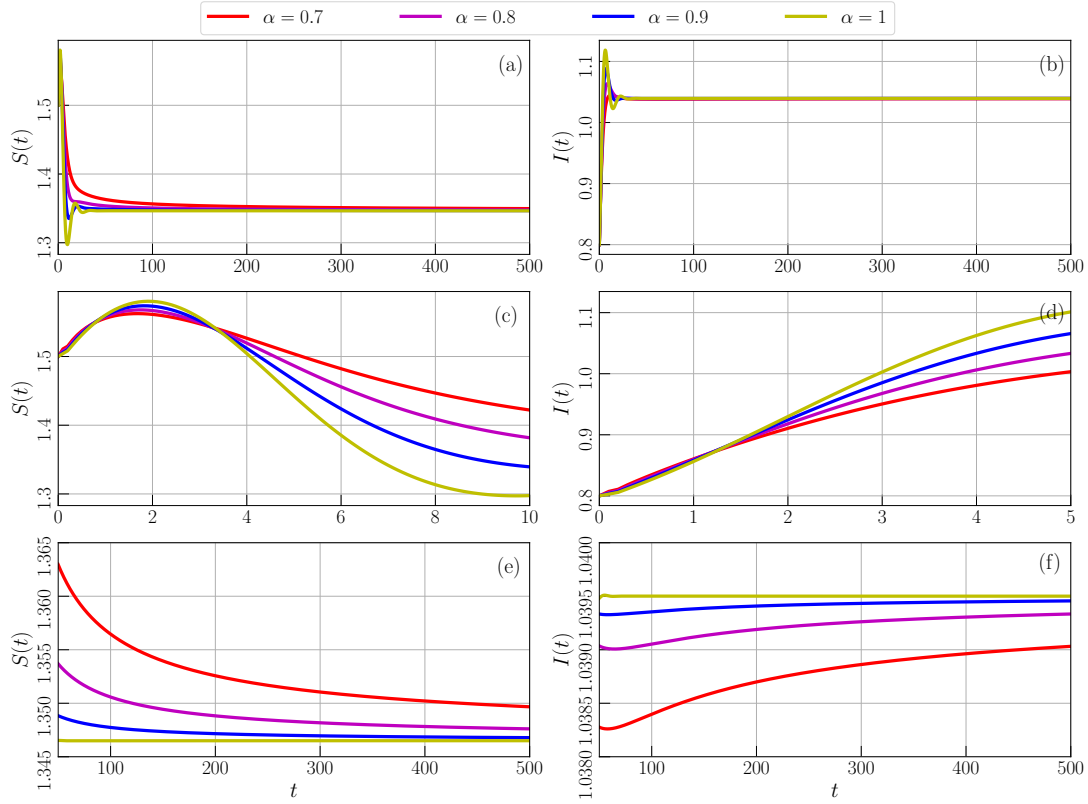


FIGURE 11. Time series of model (8) with parameter values given by eq. (20) for $\alpha = 0.7, 0.8, 0.9, 1$. **(a,b)** Time-series for $0 \leq t \leq 500$, **(c,d)** Local amplification of (a, b) around $0 \leq t \leq 10$, and **(e,f)** Local amplification of (a, b) around $100 \leq t \leq 500$

238 4. GLOBAL SENSITIVITY ANALYSIS

239 In this section, the global sensitivity analysis is studied to investigate the most influential
 240 parameters of model (8). Global sensitivity analysis is calculated using Partial Rank Coefficient
 241 Correlation (PRCC) [43], where the random data processed in PRCC is generated using Saltelli
 242 sampling [44]. Two biological components become the objective function for the PRCC namely
 243 the basic reproduction number (\mathcal{R}_0) and the population density of infected class ($I(t)$). We
 244 first investigate the most influential parameter to the basic reproduction number (\mathcal{R}_0). From
 245 eq. (12), we acquire that only r , μ , ω_1 , ω_4 , and η have the influence on the value of \mathcal{R}_0 . The
 246 birth rate and the natural death rate also can be fixed since some cases in the epidemiological
 247 model has the values of these parameters. Thus, only β , η , ω_1 , and ω_4 will be computed for
 248 PRCC. The Figure 2 is given for the results. We have $\beta = 0.763$, $\omega_1 = -0.352$, $\omega_4 = -0.33$,
 249 and $\eta = -0.277$ as the coefficient correlation such that the infection rate (β) becomes the most
 250 influential parameter to \mathcal{R}_0 and followed by ω_1 , ω_4 , and η , respectively. It shows that the
 251 infection rate (β) as the most influential parameter has a positive relationship with the basic
 252 reproduction number (\mathcal{R}_0) which means that \mathcal{R}_0 will significantly increases when β increases.
 253 The rest ω_1 , ω_4 , and η have a negative relationship with \mathcal{R}_0 which means that by reducing
 254 the value of those parameters, the basic reproduction number (\mathcal{R}_0) will increases. To show the
 255 impact of these parameters on \mathcal{R}_0 , the contour plots are also portrayed in Figure 3.

256 Next, we identify the most influential parameter to the population density of infected class
 257 ($I(t)$). Quite similar to previous work, the value of r and μ are fixed but the rest of the pa-
 258 rameters are involved to compute PRCC. PRCC values are computed for $0 \leq t \leq 50$ which
 259 is considered sufficient enough to see the convergence for each parameter through the PRCC.
 260 We portray the PRCC results in Figure 4 while the PRCC values, ranks, and the relationship
 261 between each parameter and $I(t)$ are given in Table 1. From those simulations, we conclude
 262 that the death rate of infected population due to interspecific competition between susceptible
 263 and infected classes (ω_4) become the most influential parameter to the population density ($I(t)$)
 264 followed respectively by β , η , ω_3 , ω_2 , and ω_1 . In the next section, the numerical simulations
 265 including bifurcation diagram and time-series are presented to show the impact of the infection

266 rate (β), recovery rate (η), intraspecific competition (ω_1 and ω_3), and interspecific competition
 267 (ω_2 and ω_4) to the dynamical behaviors of model (8).

268 5. NUMERICAL SIMULATIONS

269 In this section, the dynamical behaviors of model (8) including bifurcation diagram and time-
 270 series are studied numerically. To obtain the bifurcation diagram and the corresponding time-
 271 series of model (8), the predictor-corrector scheme developed by Diethelm et al. is employed
 272 [45]. Since the model does not investigate a specific epidemiological case, we use hypothetical
 273 parameters for all numerical simulations. we set the parameter values as follows.

(20)

274 $r = 0.6, \mu = 0.1, \omega_1 = 0.1, \omega_2 = 0.1, \omega_3 = 0.1, \omega_4 = 0.1, \beta = 0.4, \eta = 0.2, \text{ and } \alpha = 0.9$

275 We start our work by investigating the impact of infection rate (β) on the dynamics of model
 276 (8). The value of β is varied in the interval $0 \leq \beta \leq 1$ and we then compute the numerical
 277 solutions. To obtain the bifurcation diagram, we plot the tail of solutions for each β together
 278 with the LAS condition of \mathcal{E}_A . As result, we obtain a bifurcation diagram as in Figure 5a. When
 279 $0 \leq \beta < \beta^*, \beta^* = 0.16$, the EEP \mathcal{E}_I does not exist and Theorem 7 is satisfied which means
 280 that DFE \mathcal{E}_A is LAS. The solution is convergent to \mathcal{E}_A which indicates the population free from
 281 disease. When β passes through β^* , \mathcal{E}_A losses its stability, and unique LAS EEP \mathcal{E}_I occurs
 282 in the interior. The infectious disease becomes endemic in the population and still exists for
 283 all $t \rightarrow \infty$. From the concatenation of those biological circumstances, we conclude that forward
 284 bifurcation occurs around \mathcal{E}_A where β is the bifurcation parameter and $\beta = \beta^*$ is the bifurcation
 285 point. It is easy to examine that the bifurcation point $\beta = \beta^*$ is equal to $\mathcal{R}_0 = 1$. The dynamical
 286 behaviors are maintained for $\beta^* < \beta \leq 1$. To support these conditions, some time series are
 287 given in Figure 5b to show the convergence of solutions for different values of β .

288 Next, the impact of recover rate (η) is studied. A similar numerical scheme as the previous
 289 way is applied. To depicts the bifurcation diagram, the parameter is fixed as in eq. (20) and the
 290 recovery rate (η) is varied in interval $0 \leq \eta \leq 1$. We have Figure 6a as the result. Denote that the
 291 bifurcation does not exist for this interval. Both DFEP and EEP exist with distinct stability. The
 292 DFEP \mathcal{E}_A is a saddle point while the EEP \mathcal{E}_I is LAS which confirm the validity of Theorems 6

293 and 7. We also confirm that the EEP \mathcal{E}_I attains GAS which means that all initial conditions
 294 will go right to the EEP and the infectious disease will exist all the time. Although the disease
 295 becomes endemic, the numerical simulation shows that the value of η is directly proportional
 296 to $S(t)$ and inversely proportional to $I(t)$, see Figure 6b. This means the population density of
 297 the infected class can be reduced by increasing the recovery rate (η).

298 For the next simulation, the impact of intraspecific competition is investigated. The death
 299 rate parameters caused by intraspecific competition on susceptible and infected classes (ω_1 and
 300 ω_3) are varied in interval $[0, 1]$. It is found that forward bifurcation occurs when ω_1 is driven
 301 where the bifurcation point is given by $\omega_1^* = 0.5$, see Figure 7a. The population density of
 302 both susceptible and infected classes reduces when the death rate of $S(t)$ due to intraspecific
 303 competition increases as given by Figure 7b. Particularly, Figure 8a shows that bifurcation does
 304 not exist in interval $0 \leq \omega_1 \leq 1$ when ω_1 is varied but the dynamical behaviors show that $S(t)$
 305 increases and $I(t)$ decrease when ω_1 increase. We confirm this condition by giving time-series
 306 in Figure 8b.

307 Now, we study the impact of interspecific competition on the dynamical behaviors of model
 308 (8). Both susceptible and infected classes have died due to the existence of interspecific com-
 309 petition given by parameters ω_2 and ω_4 . By varying ω_2 and ω_4 in interval $[0, 1]$, we obtain
 310 Figures 9a and 10a as the bifurcation diagram. We find forward bifurcation driven by ω_4 which
 311 does not exist when varying ω_1 . This means, the EEP still exists and LAS for $0 \leq \omega_2 \leq 1$.
 312 The EEP will disappear via forward bifurcation and the saddle DFEP becomes LAS when ω_4
 313 crosses $\omega_4^* = 0.34$. This guarantees that the infectious disease may eliminate the disease in pop-
 314 ulation when the death rate of the infected population due to interspecific competition increases
 315 as shown in Figure 10b. Although the disease does not disappear when ω_2 is driven, we also can
 316 see in Figure 9b that by increasing ω_2 , the population density of the infected class will reduce
 317 and the susceptible class will increase.

318 Finally, the impact of memory effect (α) is investigated. The numerical simulation is given
 319 by Figure 11. For $\alpha = 0.7, 0.8, 0.9, 1$ and similar initial values, all solution converge to single
 320 equilibrium point given by $\mathcal{E}_I \approx (1.3465, 1.0395)$, see Figure 11(a,b). We then plot the local
 321 amplification to show the difference of solutions when α is varied. We find that the difference

322 lies in the convergence rate where for larger values of α , the convergence rate increase and
323 vice versa as shown in Figure 11(e,f). In the beginning, Figure 11(c,d) we show that when α
324 decrease, the population density of the infected class reduce. From a biological point of view,
325 we can say that biological memory has an impact on the density of both susceptible and infected
326 classes.

327 **6. CONCLUSION**

328 The dynamics of a fractional-order SIS-epidemic model with intraspecific and interspecific
329 competition have been studied. The validity of the model has been confirmed analytically by
330 showing the existence, uniqueness, non-negativity, and boundedness of solutions. Three equi-
331 librium points have been obtained namely the origin, the disease-free equilibrium point, and
332 the endemic equilibrium point. Both origin and disease-free equilibrium points always exist
333 while the endemic equilibrium point conditionally exists. The basic reproduction number \mathcal{R}_0
334 has been given which has a relationship with the local stability of the model. If $\mathcal{R}_0 < 1$ then the
335 disease-free equilibrium point is locally asymptotically stable and if $\mathcal{R}_0 > 1$ then the disease-
336 free equilibrium point losses its stability along with the existence of a locally asymptotically
337 stable endemic equilibrium point. The global stability conditions of equilibrium points also
338 have been found. The PRCC has been worked to investigate the most influential parameter.
339 We have successfully shown that the infection rate and the death rate of the infected population
340 due to interspecific competition becomes the most influential parameter for basic reproduction
341 number and the population density of the infected class. We then investigate the impact of sev-
342 eral parameters using numerical simulations including the infection rate, the recovery rate, the
343 intraspecific competition, the interspecific competition, and the memory effect on the dynamics
344 of the model. Bifurcation diagrams and time series have been given which show the existence
345 of forward bifurcation, the decrease of susceptible and infected classes, and the decrease of
346 convergence rate caused by the memory effect.

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350 **CONFLICT OF INTERESTS**

351 The author(s) declare that there is no conflict of interests.

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Galley-Proof Statement

18 October 2022 at 06:07 AM

[cmbn] Editor Decision #7730

Ismail Djakaria <iskar@ung.ac.id>
Kepada: CMBN Editorial Office <cmbn@scik.org>

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DYNAMICS OF SIS–EPIDEMIC MODEL WITH COMPETITION INVOLVING FRACTIONAL-ORDER DERIVATIVE WITH POWER-LAW KERNEL

ISMAIL DJAKARIA^{1,*}, HASAN S. PANIGORO², EBENEZER BONYAH³, EMLI RAHMI², WAHAB MUSA⁴

¹Magister Mathematics Education Programme, Post-Graduate, Universitas Negeri Gorontalo,
Gorontalo 96128, Indonesia

²Biomathematics Research Group, Department of Mathematics, Faculty of Mathematics and Natural Sciences,
Universitas Negeri Gorontalo, Bone Bolango 96119, Indonesia

³Department of Mathematics Education, Akenten Appiah-Menka University of Skills Training and
Entrepreneurial Development, Kumasi 00233, Ghana

⁴Department of Electrical Engineering, Universitas Negeri Gorontalo, Gorontalo 96128, Indonesia

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Abstract. Infectious disease and competition play important roles in the dynamics of a population due to their capability to increase the mortality rate for each organism. In this paper, the dynamical behaviors of a single species population are studied by considering the existence of the infectious disease, intraspecific competition, and interspecific competition. The fractional-order derivative with a power-law kernel is utilized to involve the impact of the memory effect. The population is divided into two compartments namely the susceptible class and the infected class. The existence, uniqueness, non-negativity, and boundedness of the solution are investigated to confirm the biological validity. Three types of feasible equilibrium points are identified namely the origin, the disease-free, and the endemic points. All biological conditions which present the local and global stability are investigated. The global sensitivity analysis is given to investigate the most influential parameter to the basic reproduction number and the density of each class. Some numerical simulations including bifurcation diagrams and time series are also portrayed to explore more the dynamical behaviors.

*Corresponding author

E-mail address: iskar@ung.ac.id

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1. INTRODUCTION

The spread of infectious disease still becomes a fundamental issue not only because of the existence of the population but also to maintain the balance of biological systems. Several scientific methods are developed to discover better ways to suppress and control the rate of disease infection [1]. The preferred ways for the last decades for this epidemiological problems are given by mathematical approach using a deterministic model which is considered efficacious to understand the mechanisms of disease transmission and evaluate the appropriate control strategies [2, 3, 4]. The fundamental one which has become the basis of epidemiological modeling is given by [5] which develops the continuous-time deterministic model using first-order derivative as the operator. This model is successfully developed in couple of ways such as the continuous-time single species epidemiological modeling with first-order derivative [6, 7, 8, 9], the discrete-time single species epidemiological modeling [10, 11, 12], the stochastic single-species epidemiological modeling [13, 14], and the continuous-time eco-epidemiological modeling [15, 16, 17].

Apart from those operators, several researchers prefer to use the fractional-order derivative to accomplish their problems the biological modeling. See [18, 19, 20] and references therein for some examples in epidemiological modeling. The fractional-order derivative is chosen by considering the capability of this operator to describe the current state of the biological object as the impact of all of its previous conditions which are known as the memory effect [21, 22]. In the epidemiological model, the transmission of disease may slow down and be forestalled by the susceptible population as the impact of the memory [23]. Some fractional-order derivative has been developed and successfully applied in epidemiological modeling such as the Riemann-Liouville, Caputo, Caputo-Fabrizio, and Atangana-Baleanu [24, 25, 26, 27]. From all of the given operators, the Caputo fractional-order derivative has the complete tools for dynamical analysis such as the existence and uniqueness, non-negativity and boundedness, local dynamics, global dynamics, and some bifurcation analysis. Consequently, the Caputo operator will be used in this paper where defined later in the next section.

In this work, we develop the epidemiological model based on the SIR model given by [5]. For single-species conditions, this model is only popular for the infectious diseases that appeared in the human population. In facts, infectious diseases also threaten the existence of the animal population which disturbs the balance of the ecosystem. For examples, the infectious diseases in endemic species such as Orangutans [28], Tarsius [29], Sumatran Tiger [30], and Komodo dragon [31]. Moreover, the natural behaviors of animals that endanger the existence of their populations are the intraspecific competition among them to preserve their food sources [32, 33, 34]. For these reasons, developing and investigating the dynamics of the epidemiological model by considering the impact of intraspecific competition and the memory effect are critical issues that become the novelty of our research.

The whole of this paper is organized in the following procedure: In Section 2, the mathematical modeling consists of model formulation, existence, uniqueness, non-negativity, and boundedness are given. The analytical results including the existence of equilibrium points and their local and global dynamics are completely investigated in Section 3. To show the most influential parameter of the model, the global sensitivity analysis is provided by Section 4. Some numerical simulations as well as bifurcation diagrams and time-series are presented in Section 5 to explore more about the dynamical behaviors of the model. This work ends by giving a conclusion in Section 6.

2. MATHEMATICAL MODELING

This section studies about mathematical modeling consisting of the model formulation, existence, uniqueness, non-negativity, and boundedness of solution. The mathematical model is constructed by a deterministic approach using a differential equation. We first give some assumptions to restrain the model so it does not get too complicated. We next interpret the giving assumptions to the mathematical formula using the first-order derivative as the operator. A diagram is presented to show the impact of each assumption on the flow of population density for each compartment. To involve the impact of the memory effect, the Caputo fractional-order derivative is applied to the model. For the mathematical model's validity, we show that the solution of the model always exists, unique, non-negative, and bounded.

2.1. Model Formulation. In this work, the model is constructed from a single population growth model. We first assume there exists a population in a habitat that grows proportionally to its density and bounded due to the intraspecific competition. Let $N(t)$ be the population at time t , r is the birth rate, μ is the natural death rate, and ω is the death rate as a result of competition. Thus, we have a first-order differential equation as follows.

$$(1) \quad \frac{dN}{dt} = (r - \mu)N - \omega N^2.$$

Next, we assume that the population is exposed by infectious disease. The population N is divided into two compartments namely the susceptible class (S) and infected class (I) where $N = S + I$. The susceptible class is infected by disease bilinearly with infection rate β . The competition is divided into two cases namely the intraspecific competition for each susceptible and infected class, and the interspecific competition between susceptible and infected classes. As result, the following model is received.

$$(2) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - \mu I, \end{aligned}$$

where ω_i , $i = 1, 2$ respectively denote the death rate of the susceptible population as the results of intraspecific and interspecific competitions between susceptible and susceptible classes, and susceptible and infected classes. The parameters ω_i , $i = 3, 4$ denote the death rate of the infected population as the result of competition between infected and infected classes, and susceptible and infected classes. In our works, we also assume that each organism has the capability to survive the disease. Thus, we define η as the recovery rate. Since each organism that survives from the disease has a chance to be re-infected, this type of population will be again susceptible. Finally, we have a mathematical model as follows.

$$(3) \quad \begin{aligned} \frac{dS}{dt} &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ \frac{dI}{dt} &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

All of the given assumptions and their mathematical modeling are described in Figure 1.

Now, the Caputo fractional-order derivative will be applied in order to conduct the impact of the memory effect on the population growth rate. The similar procedure is adopted from

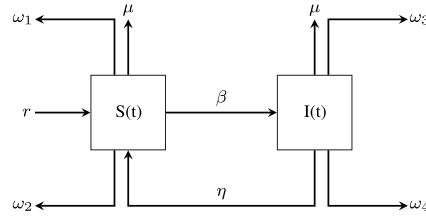


FIGURE 1. Compartment diagram of model (3)

[35]. The first-order derivatives on the left-hand side of model (3) are replaced by the Caputo fractional-order derivative defined as follows.

Definition 1. [36] Suppose $0 < \alpha \leq 1$. The Caputo fractional derivative of order $-\alpha$ is defined by

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

where $t \geq 0$, $f \in C^n([0, +\infty), \mathbb{R})$, and Γ is the Gamma function.

Applying Definition 1 to eq. (3), the following model is obtained.

$$(5) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I. \end{aligned}$$

Since the given process above makes the dimension of time at the left-hand side become t^α , some parameters need to be rescaled so that there are no differences between the time's dimensions at the left-hand side with the right-hand side of model (5). By applying time rescale to some parameters, we have the model as follows.

$$(6) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r^\alpha - \mu^\alpha)S - \omega_1^\alpha S^2 - (\omega_2^\alpha + \beta^\alpha)SI + \eta^\alpha I, \\ {}^C \mathcal{D}_t^\alpha I &= (\beta^\alpha - \omega_4^\alpha)SI - \omega_3^\alpha I^2 - (\eta^\alpha + \mu^\alpha)I. \end{aligned}$$

Let $r^\alpha = \hat{r}$, $\mu^\alpha = \hat{\mu}$, $\omega_1^\alpha = \hat{\omega}_1$, $\omega_2^\alpha = \hat{\omega}_2$, $\omega_3^\alpha = \hat{\omega}_3$, $\omega_4^\alpha = \hat{\omega}_4$, $\beta^\alpha = \hat{\beta}$, and $\eta^\alpha = \hat{\eta}$. Thus, we acquire

$$(7) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (\hat{r} - \hat{\mu})S - \hat{\omega}_1 S^2 - (\hat{\omega}_2 + \hat{\beta})SI + \hat{\eta}I, \\ {}^C \mathcal{D}_t^\alpha I &= (\hat{\beta} - \hat{\omega}_4)SI - \hat{\omega}_3 I^2 - (\hat{\eta} + \hat{\mu})I. \end{aligned}$$

For simplicity, by dropping $\hat{\cdot}$ for each parameter, we obtain the final model as follows.

$$(8) \quad \begin{aligned} {}^C \mathcal{D}_t^\alpha S &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I = F_1(N(t)), \\ {}^C \mathcal{D}_t^\alpha I &= (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I = F_2(N(t)). \end{aligned}$$

Equation (8) is the final proposed model in this paper. Although model (8) seems classic and simple, this model will be powerful to solve and investigate the existence of a closed population in a certain area without any outside intervention. Our literature review also shows that the model (8) has heretofore never been studied. Now, the basic properties of model (8) such as the existence uniqueness, non-negativity, and boundedness are investigated to confirm its biological validity.

2.2. Existence and Uniqueness. In this subsection, we will show that the model (8) has a unique solution. A similar manner given by [37] is used. Thus, the following theorem is presented to show the existence and uniqueness of the solution of model (8).

Theorem 1. *The model (8) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ has a unique solution.*

Proof. Consider model (8) with positive initial condition with $F : [0, \infty) \rightarrow \mathbb{R}^2$ where $F(N) = (F_1(N), F_2(N))$, $N \equiv N(t)$ and $\theta \equiv \{(S, I) \in \mathbb{R}_+^2 : \max\{|S|, |I|\} \leq M\}$ for sufficiently large M . Then, for any $N = (S, I)$ and $\bar{N} = (\bar{S}, \bar{I})$, $N, \bar{N} \in \theta$, we have

$$\begin{aligned} & \|F(N) - F(\bar{N})\| \\ &= |F_1(N) - F_1(\bar{N})| + |F_2(N) - F_2(\bar{N})| \\ &= \left| [(r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I] - [(r - \mu)\bar{S} - \omega_1 \bar{S}^2 - (\omega_2 + \beta)\bar{S}\bar{I} + \eta \bar{I}] \right| + \\ & \quad \left| [(\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I] - [(\beta - \omega_4)\bar{S}\bar{I} - \omega_3 \bar{I}^2 - (\eta + \mu)\bar{I}] \right| \\ &\leq (r + \mu) |S - \bar{S}| + \omega_1 |S^2 - \bar{S}^2| + (\omega_2 + \beta) |SI - \bar{S}\bar{I}| + \eta |I - \bar{I}| + (\beta + \omega_4) |SI - \bar{S}\bar{I}| \\ & \quad + \omega_3 |I^2 - \bar{I}^2| + (\eta + \mu) |I - \bar{I}| \\ &= (r + \mu) |S - \bar{S}| + \omega_1 |(S + \bar{S})(S - \bar{S})| + (\omega_2 + \omega_4 + 2\beta) |I(S - \bar{S}) + \bar{S}(I - \bar{I})| \\ & \quad + (2\eta + \mu) |I - \bar{I}| + \omega_3 |(I + \bar{I})(I - \bar{I})| \end{aligned}$$

$$\begin{aligned}
&\leq (r + \mu) |S - \bar{S}| + 2\omega_1 M |S - \bar{S}| + (\omega_2 + \omega_4 + 2\beta) M |S - \bar{S}| \\
&\quad + (\omega_2 + \omega_4 + 2\beta) M |I - \bar{I}| + (2\eta + \mu) |I - \bar{I}| + 2\omega_3 M |I - \bar{I}| \\
&= [(r + \mu) + 2\omega_1 M + (\omega_2 + \omega_4 + 2\beta) M] |S - \bar{S}| \\
&\quad + [(\omega_2 + \omega_4 + 2\beta) M + (2\eta + \mu) + 2\omega_3 M] |I - \bar{I}| \\
&\leq L \|N - \bar{N}\|,
\end{aligned}$$

where $L = (\omega_2 + \omega_4 + 2\beta) M + \mu + \max\{r + 2\omega_1 M, 2(\eta + \omega_3 M)\}$. Therefore, $F(N)$ satisfies the Lipschitz condition. Obeying Lemma 5 in [38], we conclude that model (8) with positive initial condition has a unique solution. \square

2.3. Non-negativity and Boundedness. The non-negativity and boundedness properties of the solutions of the model (8) are given in the following theorem.

Theorem 2. *All solution of the model (8), which start in $\mathbb{R}_+^2 := \{(S, I) \mid S \geq 0, I \geq 0, (S, I) \in \mathbb{R}^2\}$ are uniformly bounded and non-negative.*

Proof. To prove the boundedness of the solutions of the model (8), the same approach of [38] is adopted. Let consider the function $N = S + I$. Then,

$$\begin{aligned}
{}^C \mathcal{D}_t^\alpha N &= {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \beta)SI + \eta I + (\beta - \omega_4)SI - \omega_3 I^2 - (\eta + \mu)I \\
&= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I.
\end{aligned}$$

Hence, for each $\mu > 0$,

$$\begin{aligned}
{}^C \mathcal{D}_t^\alpha N + \mu N &= (r - \mu)S - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I + \mu S + \mu I \\
&= rS - \omega_1 S^2 - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&= -\omega_1 \left(S - \frac{r}{2\omega_1} \right)^2 + \frac{r^2}{4\omega_1} - (\omega_2 + \omega_4)SI - \omega_3 I^2 \\
&\leq \frac{r^2}{4\omega_1}
\end{aligned}$$

By using the comparison theorem in [39], we obtain $N(t) \leq N(0)E_\alpha(-\mu t^\alpha) + \frac{r^2}{4\omega_1} t^\alpha E_{\alpha, \alpha+1}(-\mu t^\alpha)$, where E_α and $E_{\alpha, \alpha+1}$ is the Mittag-Leffler function with one and two parameters. According to Lemma 5 and Corollary 6 in [39], we have $N(t) \leq \frac{r^2}{4\mu\omega_1}$, as $t \rightarrow \infty$. Therefore, all solutions of model (8) starting in \mathbb{R}_+^2 are uniformly bounded in the region Φ , where $\Phi = \left\{ (S, I) \in \mathbb{R}_+^2 : S + I \leq \frac{r^2}{4\mu\omega_1} + \varepsilon, \varepsilon > 0 \right\}$. Next, we prove that all solutions of model (8) are non-negative. By model (8), we have ${}^C \mathcal{D}_t^\alpha S|_{S=0} = \eta I \geq 0$ and ${}^C \mathcal{D}_t^\alpha I|_{I=0} = 0 \geq 0$. Based on Lemmas 5 and 6 in [40], we conclude that the solutions of model (8) are non-negative. \square

3. ANALYTICAL RESULTS

In this section, the dynamics of model (8) are shown analytically including the existence of equilibrium points, and their local and global stability.

3.1. Existence of Equilibrium Points. To find the equilibrium points of model (8), we must have

$$(9) \quad [(r - \mu) - \omega_1 S - (\omega_2 + \beta)I]S + \eta I = 0,$$

$$(10) \quad [(\beta - \omega_4)S - \omega_3 I - (\eta + \mu)]I = 0.$$

If $I = 0$ is substituted to (9), we obtain

$$(11) \quad [(r - \mu) - \omega_1 S]S = 0.$$

From eq. (11), we get $S = 0$ and $S = \frac{r - \mu}{\omega_1}$. Thus, we have two equilibrium points here namely $\mathcal{E}_0 = (0, 0)$, and $\mathcal{E}_A = \left(\frac{r - \mu}{\omega_1}, 0 \right)$. The equilibrium point \mathcal{E}_0 is called the origin point which represents the extinction of both susceptible and infected populations. Since $\mathcal{E}_0 \in \mathbb{R}_+^2$, this equilibrium point always exists. Furthermore, the equilibrium point \mathcal{E}_A is called the disease-free equilibrium point (DFEP) which describes the condition where the infectious disease does not exist anymore in the population. According to the biological condition, it is natural that the birth rate r is greater than its death rate μ . By assuming $r > \mu$, the origin point $\mathcal{E}_A \in \mathbb{R}_+^2$ also always exists. By simple calculation, we also obtain the basic reproduction number \mathcal{R}_0 given by

$$(12) \quad \mathcal{R}_0 = \frac{(r - \mu)\beta}{(r - \mu)\omega_4 + (\eta + \mu)\omega_1}.$$

The basic reproduction number is utilized to show the dynamical behavior of each equilibrium point and to describe whether the infectious disease becomes endemic or not. Since $r > \mu$, the value of \mathcal{R}_0 is always positive. Now, let's concern the eq. (9) and (10). By solving eq. (10), we attain

$$(13) \quad S = \frac{\omega_3 I + (\eta + \mu)}{\beta - \omega_4}.$$

If we substitute eq. (13) to (9), the following polynomial equation holds.

$$(14) \quad k_1 I^2 + k_2 I + k_3 = 0,$$

where

$$\begin{aligned} k_1 &= ((\beta - \omega_4)(\beta + \omega_2) + \omega_1 \omega_3) \omega_3, \\ k_2 &= (\beta - \omega_4)((\beta + \omega_2)\mu + (\omega_2 + \omega_4)\eta - (r - \mu)\omega_3) + 2(\eta + \mu)\omega_1 \omega_3, \\ k_3 &= \frac{(1 - \mathcal{R}_0)(r - \mu)(\eta + \mu)\beta}{\mathcal{R}_0}. \end{aligned}$$

Therefore, we acquire the endemic point (EEP)

$$(15) \quad \mathcal{E}_I = \left(\frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}, \bar{\gamma} \right),$$

where $\bar{\gamma}$ is the positive root of polynomial equation (14). From (15), we find that $\beta > \omega_4$ must be fulfilled so that $\mathcal{E}_I \in \mathbb{R}_+^2$. Moreover, EEP exists if $\bar{\gamma} > 0$. From eq. (14), we have k_1 is always positive. Thus, the value of the $\bar{\gamma}$ depends on k_2 and k_3 . Furthermore, eq. (14) has real number roots if $k_2^2 \geq 4k_1 k_3$. By applying simple algebra, if $k_3 > 0$ and $k_2 < 0$ then we have two positive roots of eq. (14), if $k_3 > 0$ and $k_2 > 0$ then we do not have any positive roots of eq. (14), and if $k_3 < 0$ then we have a positive root of eq. (14). Finally, we have the following theorem.

Theorem 3. *Let $\beta > \omega_4$. The existence of EEP \mathcal{E}_I is shown by the following statement.*

- (i) *If $k_2^2 < 4k_1 k_3$ then \mathcal{E}_I does not exist.*
- (ii) *If $k_2^2 = 4k_1 k_3$ and*
 - (ii.i) *if $k_2 > 0$ then \mathcal{E}_I does not exist.*
 - (ii.ii) *if $k_2 < 0$ then \mathcal{E}_I exists and unique.*
- (iii) *If $k_2^2 > 4k_1 k_3$ and*

(iii.i) if $k_3 > 0$ and $k_2 < 0$ then we have a pair of \mathcal{E}_1 .

(iii.ii) if $k_3 > 0$ and $k_2 > 0$ then \mathcal{E}_1 does not exist.

(iii.iii) if $k_3 < 0$ then \mathcal{E}_1 exists and unique.

Denote that $k_2^2 > 4k_1k_3$ is always satisfied and $k_3 < 0$ for $\mathcal{R}_0 > 1$, then the following lemma holds.

Lemma 4. *EEP \mathcal{E}_1 exists and unique if $\mathcal{R}_0 > 1$.*

3.2. Local Dynamics. The local dynamics of model (8) are obtained by applying the Matignon condition which is defined as follows.

Theorem 5. [Matignon condition [36]] *An equilibrium point \vec{x}^* is locally asymptotically stable (LAS) if all eigenvalues λ_j of the Jacobian matrix $J = \frac{\partial \vec{f}}{\partial \vec{x}}$ at \vec{x}^* satisfy $|\arg(\lambda_j)| > \frac{\alpha\pi}{2}$. If there exists at least one eigenvalue satisfy $|\arg(\lambda_k)| > \frac{\alpha\pi}{2}$ while $|\arg(\lambda_l)| < \frac{\alpha\pi}{2}$, $k \neq l$, then \vec{x}^* is a saddle-point.*

Therefore, to study the local dynamics of model (8), we first compute its Jacobian matrix at the point (S, I) which gives

$$(16) \quad \mathcal{J}(S, I) = \begin{bmatrix} (r - \mu) - 2\omega_1 S - (\omega_2 + \beta)I & -(\omega_2 + \beta)S + \eta \\ (\beta - \omega_4)I & (\beta - \omega_4)S - 2\omega_3 I - (\eta + \mu) \end{bmatrix}.$$

Obeying Theorem 5 and using Jacobian matrix (16), we discuss the local stability for each equilibrium point in the next subsection.

3.3. Dynamical behavior around \mathcal{E}_0 . LAS condition of \mathcal{E}_0 is obtained by identifying the eigenvalues of the Jacobian matrix (16) at the point $(S, I) = (0, 0)$. We receive

$$\mathcal{J}(S, I)|_{\mathcal{E}_0} = \begin{bmatrix} r - \mu & \eta \\ 0 & -(\eta + \mu) \end{bmatrix}.$$

Therefore, we have $\lambda_1 = r - \mu$ and $\lambda_2 = -(\eta + \mu)$. Since $r > \mu$ and $\lambda_2 < 0$, we have $|\arg(\lambda_1)| = 0 < \frac{\alpha\pi}{2}$ and $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$. According to Theorem 5, the following theorem holds.

Theorem 6. *The origin point \mathcal{E}_0 is always a saddle point.*

3.4. Dynamical behavior around \mathcal{E}_A . For $(x, y) = \left(\frac{r-\mu}{\omega_1}, 0\right)$, the Jacobian matrix (16) becomes

$$\mathcal{J}(S, I)|_{\mathcal{E}_A} = \begin{bmatrix} -(r-\mu) & \eta - \frac{(\omega_2+\beta)(r-\mu)}{\omega_1} \\ 0 & \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0} \end{bmatrix},$$

which gives a pair of eigenvalues $\lambda_1 = -(r-\mu)$ and $\lambda_2 = \frac{(\mathcal{R}_0-1)(r-\mu)\beta}{\omega_1\mathcal{R}_0}$. Denote $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$ as the impact of $\lambda_1 < 0$. Hence, the sign of λ_2 takes the role in describing local dynamics around \mathcal{E}_A . To obtain $|\arg(\lambda_2)| = \pi > \frac{\alpha\pi}{2}$, we need $\lambda_2 < 0$ which is fulfilled if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ then $|\arg(\lambda_2)| = 0 < \frac{\alpha\pi}{2}$. Following the Matignon condition given in Theorem 5, the following theorem is successfully attained.

Theorem 7. *If $\mathcal{R}_0 < 1$ then \mathcal{E}_A is LAS and a saddle point if $\mathcal{R}_0 > 1$.*

3.5. Dynamical behavior around \mathcal{E}_I . To identify the local stability of \mathcal{E}_I , we first compute the Jacobian matrix (16) evaluated at \mathcal{E}_I . We generate

$$(17) \quad \mathcal{J}(S, I)|_{\mathcal{E}_I} = \begin{bmatrix} -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu}\right] & -\frac{(\omega_2+\beta)(\omega_3\bar{\gamma}+\eta+\mu)}{\beta-\omega_4} + \eta \\ (\beta-\omega_4)\bar{\gamma} & -\omega_3\bar{\gamma} \end{bmatrix}.$$

The eigenvalues of (17) are given by $\lambda_1 = \frac{1}{2} \left(\xi_1 + \sqrt{\xi_1^2 - 4\xi_2} \right)$ and $\lambda_2 = \frac{1}{2} \left(\xi_1 - \sqrt{\xi_1^2 - 4\xi_2} \right)$ where

$$\begin{aligned} \xi_1 &= -\left[\frac{(\omega_3\bar{\gamma}+\eta+\mu)\omega_1}{\beta-\omega_4} + \frac{(\beta-\omega_4)\eta\bar{\gamma}}{\omega_3\bar{\gamma}+\eta+\mu} + \omega_3\bar{\gamma} \right], \\ \xi_2 &= \left[\left(\frac{\omega_1\omega_3}{\beta-\omega_4} + \omega_2 + \beta \right) (\omega_3\bar{\gamma} + \eta + \mu) + \left(\frac{\omega_3\bar{\gamma}}{\omega_3\bar{\gamma} + \eta + \mu} + 1 \right) (\beta - \omega_4)\eta \right] \bar{\gamma}. \end{aligned}$$

It is easy to proof that $\xi_1 < 0$ and $\xi_2 > 0$ since $\beta > \omega_4$ becomes the existence condition. As the impact, $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$, $i = 1, 2$ and hence the LAS always hold for EEP. Thus, the following theorem holds.

Theorem 8. *EEP \mathcal{E}_I is always LAS.*

3.6. Global Dynamics. In this subsection, the global dynamics of model (8) are studied. The biological conditions of equilibrium points are investigated so that those points are globally asymptotically stable (GAS). Since the origin is always a saddle point, we focus on studying GAS conditions for DFEP and EEP. The next two theorems are given for the global dynamics.

Theorem 9. DFEP \mathcal{E}_A is GAS if $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$.

Proof. We define a positive Lyapunov function as follows.

$$(18) \quad \mathcal{V}_A(S, I) = \left(S - \frac{r - \mu}{\omega_1} - \frac{r - \mu}{\omega_1} \ln \frac{\omega_1 S}{r - \mu} \right) + I.$$

If we calculate the Caputo fractional derivative of $\mathcal{V}_A(S, I)$ along the solution of model (8) and use Lemma 3.1 in [41], we get

$$\begin{aligned} & {}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \\ &= \left(\frac{S - \frac{r - \mu}{\omega_1}}{S} \right) {}^C \mathcal{D}_t^\alpha S + {}^C \mathcal{D}_t^\alpha I \\ &= -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 + \frac{(r - \mu)(\omega_2 + \beta)I}{\omega_1} - \frac{(r - \mu)\eta I}{\omega_1 S} - (\omega_2 + \omega_4)SI - \omega_3 I^2 - \mu I \\ &\leq -\omega_1 \left(S - \frac{r - \mu}{\omega_1} \right)^2 - \left(\mu - \frac{(\omega_2 + \beta)r}{\omega_1} \right) I \end{aligned}$$

Since $\omega_1 > \frac{(\omega_2 + \beta)r}{\mu}$, we have ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$, and ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$ only when $(S, I) = \left(\frac{r - \mu}{\omega_1}, 0 \right)$. This means that the singleton $\{\mathcal{E}_A\}$ is the only invariant set where ${}^C \mathcal{D}_t^\alpha \mathcal{V}_A(S, I) = 0$. By Lemma 4.6 in [42], we can conclude that every solution of model (8) tends to DFEP \mathcal{E}_A . □

Theorem 10. EEP \mathcal{E}_I is GAS if $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$.

Proof. We first define $\vartheta = \frac{\omega_3 \bar{\gamma} + (\eta + \mu)}{\beta - \omega_4}$ and hence $\mathcal{E}_I = (\vartheta, \bar{\gamma})$. Now, a positive Lyapunov function is presented as follows.

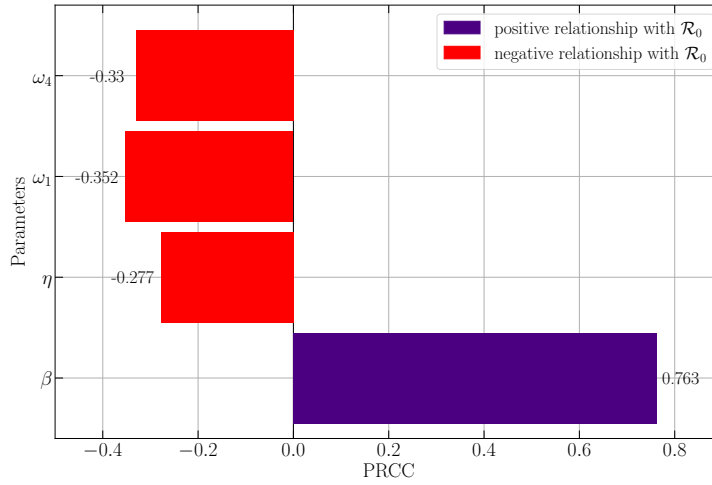
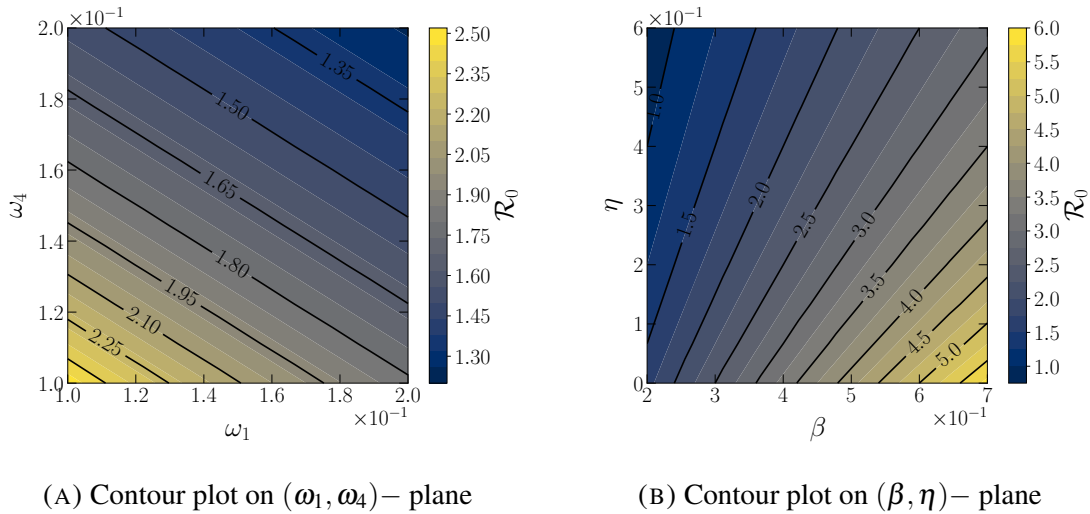
$$(19) \quad \mathcal{V}_I(S, I) = \left(S - \vartheta - \vartheta \ln \frac{S}{\vartheta} \right) + \left(I - \bar{\gamma} - \bar{\gamma} \ln \frac{S}{\bar{\gamma}} \right)$$

Following Lemma 3.1 in [41], we reach

$$\begin{aligned} & {}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \\ &= \left(\frac{S - \vartheta}{S} \right) {}^C \mathcal{D}_t^\alpha S + \left(\frac{I - \bar{\gamma}}{I} \right) {}^C \mathcal{D}_t^\alpha I \\ &= (S - S^*) \left((r - \mu) - \omega_1 S - (\omega_2 + \beta)I + \frac{\eta I}{S} \right) + (I - \bar{\gamma}) ((\beta - \omega_4)S - \omega_3 I - (\eta + \mu)) \end{aligned}$$

$$\begin{aligned}
 &= -\omega_1 (S - \vartheta)^2 - \omega_3 (I - \bar{\gamma})^2 - (\omega_2 + \omega_4) (S - S^*) (I - \bar{\gamma}) \\
 &\leq -\left(\omega_1 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta}\right)\right) (S - \vartheta)^2 - \left(\omega_3 - \left(\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta}\right)\right) (I - \bar{\gamma})^2
 \end{aligned}$$

Denote that ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) \leq 0$ for all $(S, I) \in \mathbb{R}_+^2$ as a result of $\frac{\omega_2}{2} + \frac{\omega_4}{2} + \frac{\eta}{2\vartheta} < \min\{\omega_1, \omega_3\}$. We also have that ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$ only when $(S, I) = (\vartheta, \bar{\gamma})$. Therefore, the singleton $\{\mathcal{E}_I\}$ is the only invariant set where ${}^C \mathcal{D}_t^\alpha \mathcal{V}_I(S, I) = 0$. Obeying Lemma 4.6 in [42], every solution of model (8) tends to EEP \mathcal{E}_I . \square


 FIGURE 2. PRCC results for the parameters of \mathcal{R}_0

 FIGURE 3. Contour plots for the parameters respect to \mathcal{R}_0

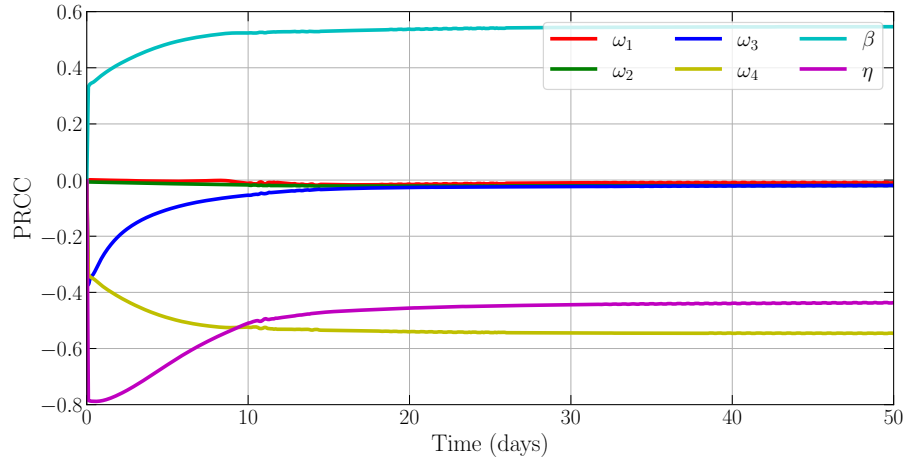
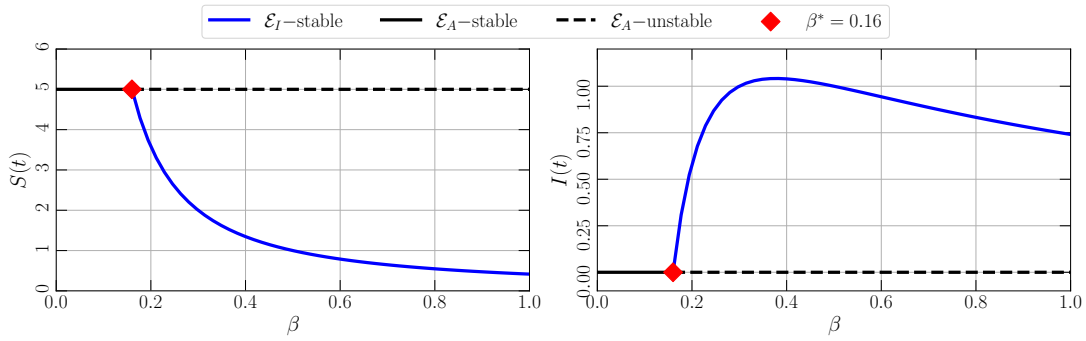
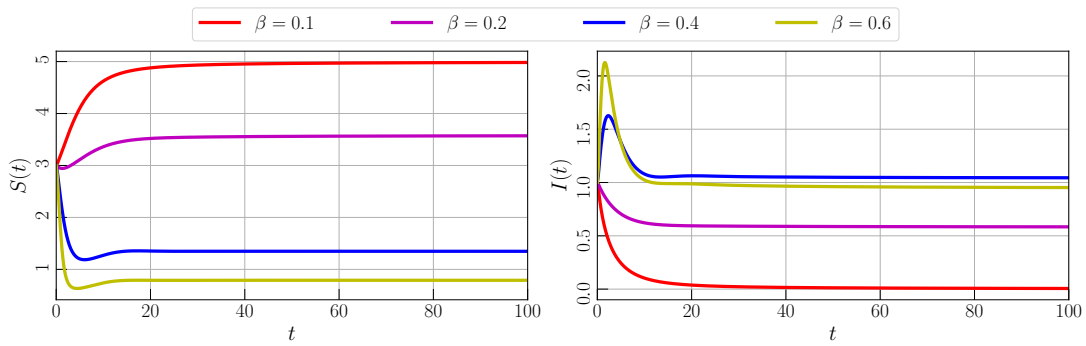
FIGURE 4. PRCC results for the parameters of $I(t)$

TABLE 1. PRCC results in respect to the population density of infected class

Parameter	Description	PRCC	Rank	Relationship with $I(t)$
ω_1	The death rate of susceptible population due to the intraspecific competition	-0.00851	6	Negative relationship
ω_2	The death rate of susceptible population due to the interspecific competition	-0.01938	5	Negative relationship
ω_3	The death rate of infected population due to the intraspecific competition	-0.01990	4	Negative relationship
ω_4	The death rate of infected population due to the interspecific competition	-0.54635	1	Negative relationship
β	The infection rate	0.54631	2	Positive relationship
η	The recovery rate	-0.43606	3	Negative relationship



(A) Bifurcation diagram driven by β in interval $0 \leq \beta \leq 1$



(B) Time-series for $\beta = 0.1, 0.2, 0.4,$ and 0.6

FIGURE 5. Bifurcation diagram and times-series of model (8) driven by the infection rate (β) with parameter values given by eq. (20)

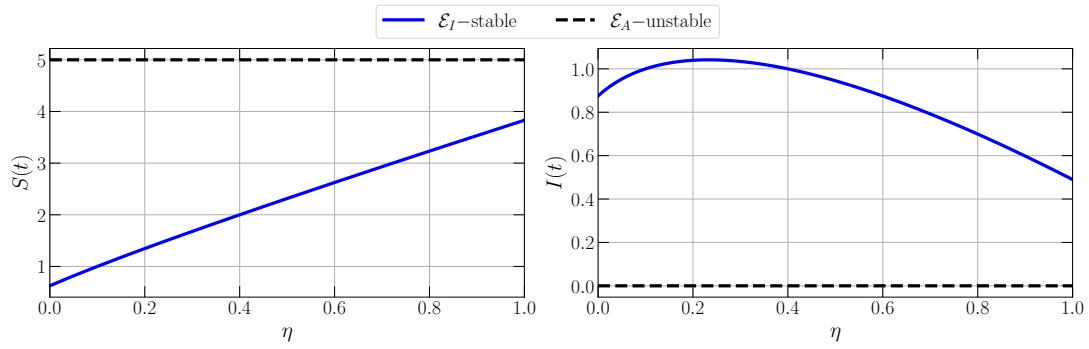
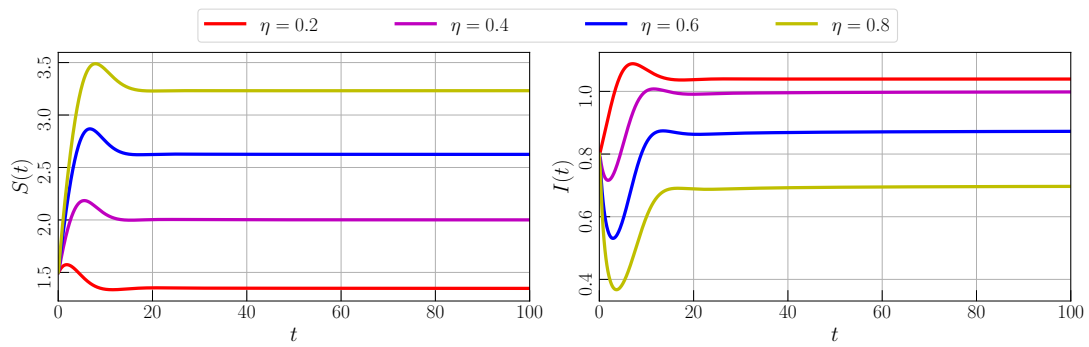
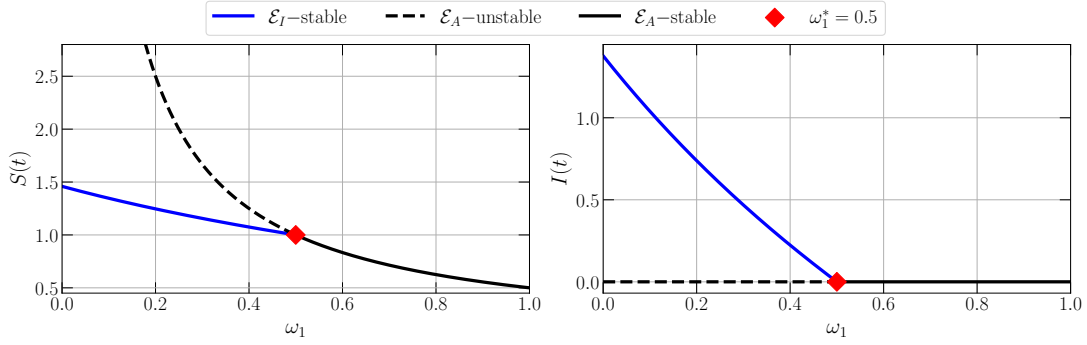
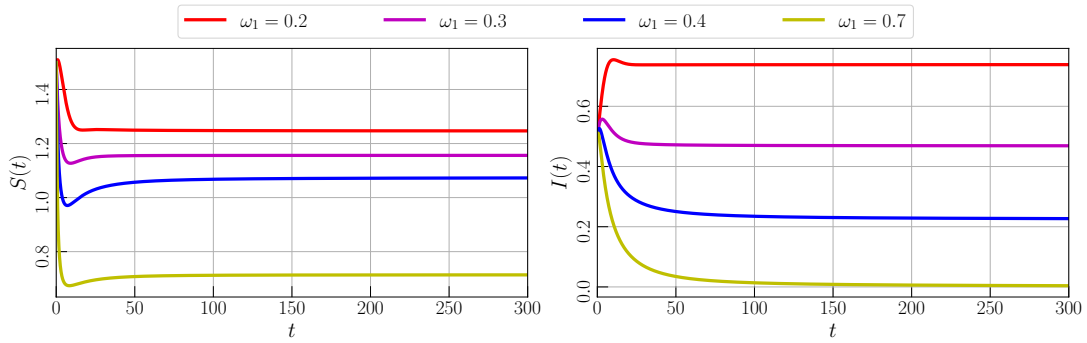
(A) Bifurcation diagram driven by η in interval $0 \leq \eta \leq 1$ (B) Time-series for $\eta = 0.2, 0.4, 0.6,$ and 0.8

FIGURE 6. Bifurcation diagram and times-series of model (8) driven by the recovery rate (η) with parameter values given by eq. (20)



(A) Bifurcation diagram driven by ω_1 in interval $0 \leq \omega_1 \leq 1$



(B) Time-series for $\omega_1 = 0.2, 0.3, 0.4,$ and 0.7

FIGURE 7. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to intraspecific competition (ω_1) with parameter values given by eq. (20)

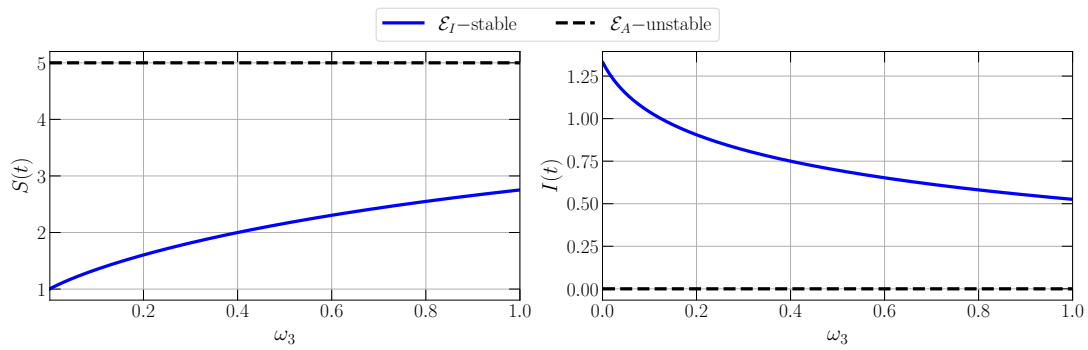
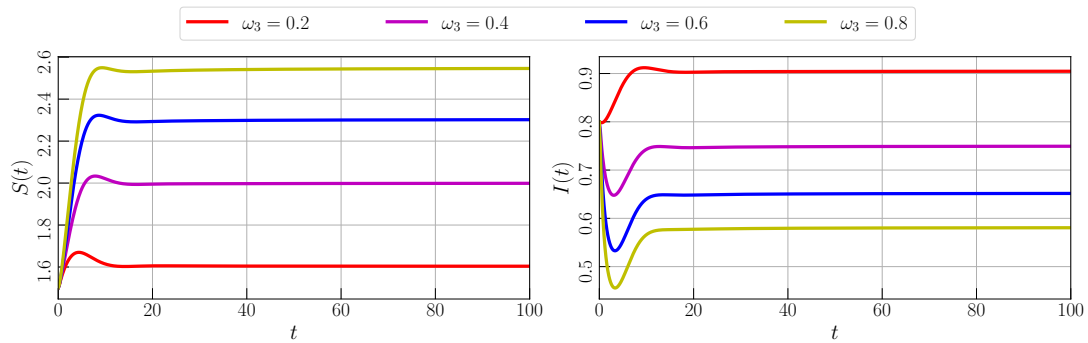
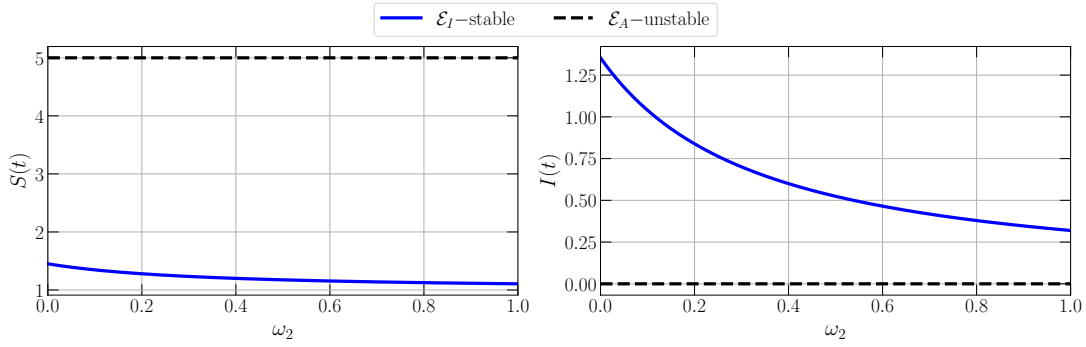
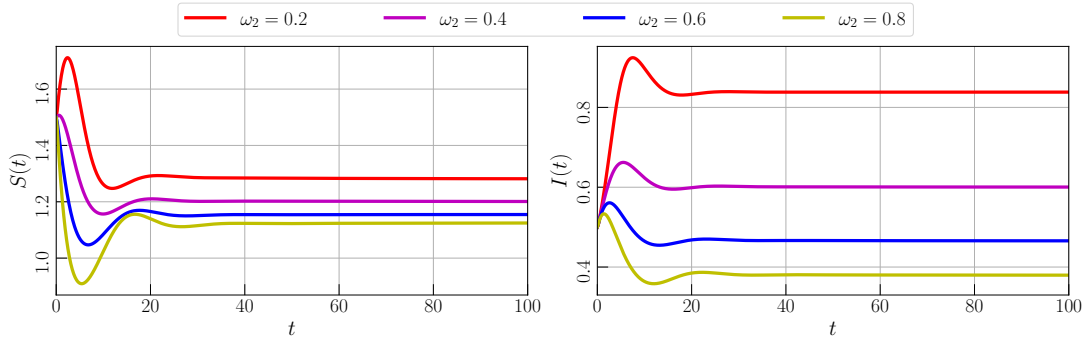
(A) Bifurcation diagram driven by ω_3 in interval $0 \leq \omega_3 \leq 1$ (B) Time-series for $\omega_3 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 8. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to intraspecific competition (ω_3) with parameter values given by eq. (20)



(A) Bifurcation diagram driven by ω_2 in interval $0 \leq \omega_2 \leq 1$



(B) Time-series for $\omega_2 = 0.2, 0.4, 0.6,$ and 0.8

FIGURE 9. Bifurcation diagram and times-series of model (8) driven by the death rate of susceptible population due to interspecific competition (ω_2) with parameter values given by eq. (20)

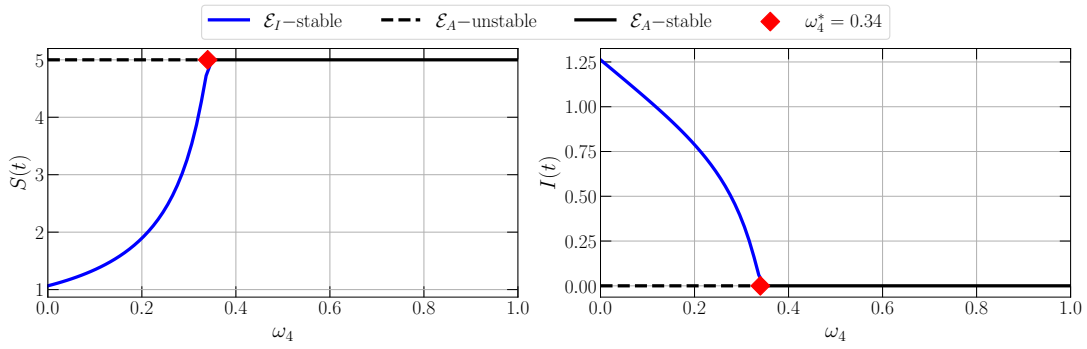
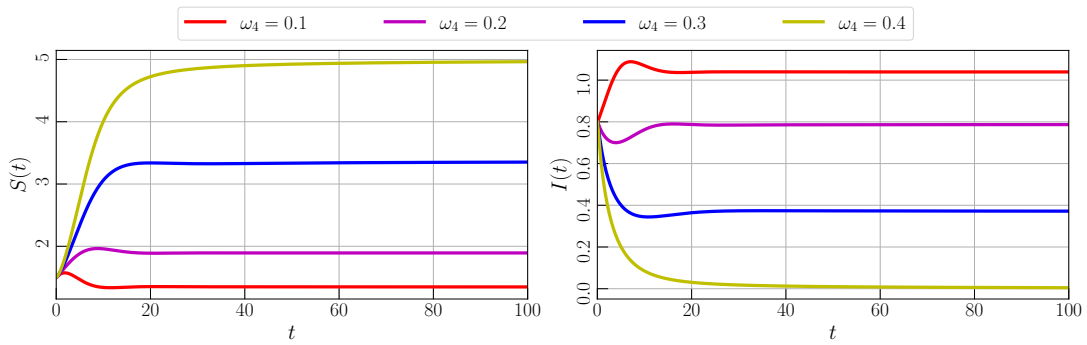
(A) Bifurcation diagram driven by ω_4 in interval $0 \leq \omega_4 \leq 1$ (B) Time-series for $\omega_4 = 0.2, 0.4, 0.6$, and 0.8

FIGURE 10. Bifurcation diagram and times-series of model (8) driven by the death rate of infected population due to interspecific competition (ω_4) with parameter values given by eq. (20)

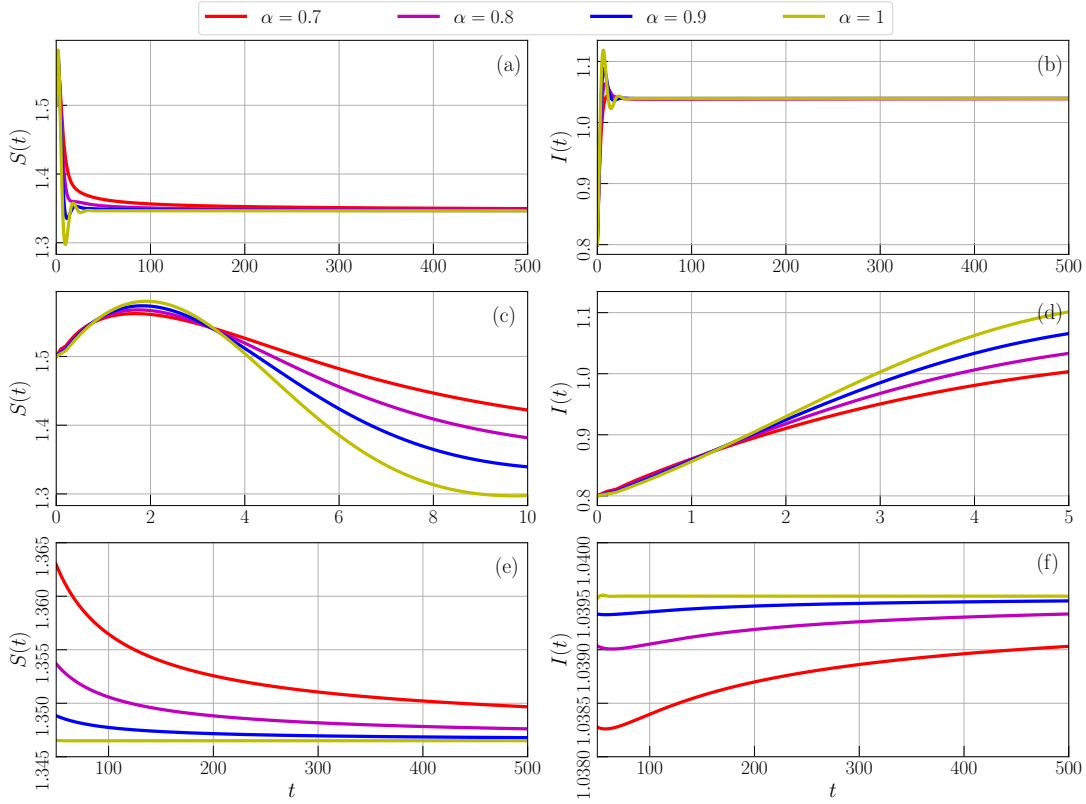


FIGURE 11. Time series of model (8) with parameter values given by eq. (20) for $\alpha = 0.7, 0.8, 0.9, 1$. **(a,b)** Time-series for $0 \leq t \leq 500$, **(c,d)** Local amplification of (a, b) around $0 \leq t \leq 10$, and **(e,f)** Local amplification of (a, b) around $100 \leq t \leq 500$

4. GLOBAL SENSITIVITY ANALYSIS

In this section, the global sensitivity analysis is studied to investigate the most influential parameters of model (8). Global sensitivity analysis is calculated using Partial Rank Coefficient Correlation (PRCC) [43], where the random data processed in PRCC is generated using Saltelli sampling [44]. Two biological components become the objective function for the PRCC namely the basic reproduction number (\mathcal{R}_0) and the population density of infected class ($I(t)$). We first investigate the most influential parameter to the basic reproduction number (\mathcal{R}_0). From eq. (12), we acquire that only r , μ , ω_1 , ω_4 , and η have the influence on the value of \mathcal{R}_0 . The birth rate and the natural death rate also can be fixed since some cases in the epidemiological model has the values of these parameters. Thus, only β , η , ω_1 , and ω_4 will be computed for PRCC. The Figure 2 is given for the results. We have $\beta = 0.763$, $\omega_1 = -0.352$, $\omega_4 = -0.33$, and $\eta = -0.277$ as the coefficient correlation such that the infection rate (β) becomes the most influential parameter to \mathcal{R}_0 and followed by ω_1 , ω_4 , and η , respectively. It shows that the infection rate (β) as the most influential parameter has a positive relationship with the basic reproduction number (\mathcal{R}_0) which means that \mathcal{R}_0 will significantly increases when β increases. The rest ω_1 , ω_4 , and η have a negative relationship with \mathcal{R}_0 which means that by reducing the value of those parameters, the basic reproduction number (\mathcal{R}_0) will increases. To show the impact of these parameters on \mathcal{R}_0 , the contour plots are also portrayed in Figure 3.

Next, we identify the most influential parameter to the population density of infected class ($I(t)$). Quite similar to previous work, the value of r and μ are fixed but the rest of the parameters are involved to compute PRCC. PRCC values are computed for $0 \leq t \leq 50$ which is considered sufficient enough to see the convergence for each parameter through the PRCC. We portray the PRCC results in Figure 4 while the PRCC values, ranks, and the relationship between each parameter and $I(t)$ are given in Table 1. From those simulations, we conclude that the death rate of infected population due to interspecific competition between susceptible and infected classes (ω_4) become the most influential parameter to the population density ($I(t)$) followed respectively by β , η , ω_3 , ω_2 , and ω_1 . In the next section, the numerical simulations including bifurcation diagram and time-series are presented to show the impact of the infection

rate (β), recovery rate (η), intraspecific competition (ω_1 and ω_3), and interspecific competition (ω_2 and ω_4) to the dynamical behaviors of model (8).

5. NUMERICAL SIMULATIONS

In this section, the dynamical behaviors of model (8) including bifurcation diagram and time-series are studied numerically. To obtain the bifurcation diagram and the corresponding time-series of model (8), the predictor-corrector scheme developed by Diethelm et al. is employed [45]. Since the model does not investigate a specific epidemiological case, we use hypothetical parameters for all numerical simulations. we set the parameter values as follows.

(20)

$$r = 0.6, \mu = 0.1, \omega_1 = 0.1, \omega_2 = 0.1, \omega_3 = 0.1, \omega_4 = 0.1, \beta = 0.4, \eta = 0.2, \text{ and } \alpha = 0.9$$

We start our work by investigating the impact of infection rate (β) on the dynamics of model (8). The value of β is varied in the interval $0 \leq \beta \leq 1$ and we then compute the numerical solutions. To obtain the bifurcation diagram, we plot the tail of solutions for each β together with the LAS condition of \mathcal{E}_A . As result, we obtain a bifurcation diagram as in Figure 5a. When $0 \leq \beta < \beta^*$, $\beta^* = 0.16$, the EEP \mathcal{E}_I does not exist and Theorem 7 is satisfied which means that DFE \mathcal{E}_A is LAS. The solution is convergent to \mathcal{E}_A which indicates the population free from disease. When β passes through β^* , \mathcal{E}_A losses its stability, and unique LAS EEP \mathcal{E}_I occurs in the interior. The infectious disease becomes endemic in the population and still exists for all $t \rightarrow \infty$. From the concatenation of those biological circumstances, we conclude that forward bifurcation occurs around \mathcal{E}_A where β is the bifurcation parameter and $\beta = \beta^*$ is the bifurcation point. It is easy to examine that the bifurcation point $\beta = \beta^*$ is equal to $\mathcal{R}_0 = 1$. The dynamical behaviors are maintained for $\beta^* < \beta \leq 1$. To support these conditions, some time series are given in Figure 5b to show the convergence of solutions for different values of β .

Next, the impact of recover rate (η) is studied. A similar numerical scheme as the previous way is applied. To depicts the bifurcation diagram, the parameter is fixed as in eq. (20) and the recovery rate (η) is varied in interval $0 \leq \eta \leq 1$. We have Figure 6a as the result. Denote that the bifurcation does not exist for this interval. Both DFEP and EEP exist with distinct stability. The DFEP \mathcal{E}_A is a saddle point while the EEP \mathcal{E}_I is LAS which confirm the validity of Theorems 6

and 7. We also confirm that the EEP \mathcal{E}_I attains GAS which means that all initial conditions will go right to the EEP and the infectious disease will exist all the time. Although the disease becomes endemic, the numerical simulation shows that the value of η is directly proportional to $S(t)$ and inversely proportional to $I(t)$, see Figure 6b. This means the population density of the infected class can be reduced by increasing the recovery rate (η).

For the next simulation, the impact of intraspecific competition is investigated. The death rate parameters caused by intraspecific competition on susceptible and infected classes (ω_1 and ω_3) are varied in interval $[0, 1]$. It is found that forward bifurcation occurs when ω_1 is driven where the bifurcation point is given by $\omega_1^* = 0.5$, see Figure 7a. The population density of both susceptible and infected classes reduces when the death rate of $S(t)$ due to intraspecific competition increases as given by Figure 7b. Particularly, Figure 8a shows that bifurcation does not exist in interval $0 \leq \omega_1 \leq 1$ when ω_1 is varied but the dynamical behaviors show that $S(t)$ increases and $I(t)$ decrease when ω_1 increase. We confirm this condition by giving time-series in Figure 8b.

Now, we study the impact of interspecific competition on the dynamical behaviors of model (8). Both susceptible and infected classes have died due to the existence of interspecific competition given by parameters ω_2 and ω_4 . By varying ω_2 and ω_4 in interval $[0, 1]$, we obtain Figures 9a and 10a as the bifurcation diagram. We find forward bifurcation driven by ω_4 which does not exist when varying ω_1 . This means, the EEP still exists and LAS for $0 \leq \omega_2 \leq 1$. The EEP will disappear via forward bifurcation and the saddle DFEP becomes LAS when ω_4 crosses $\omega_4^* = 0.34$. This guarantees that the infectious disease may eliminate the disease in population when the death rate of the infected population due to interspecific competition increases as shown in Figure 10b. Although the disease does not disappear when ω_2 is driven, we also can see in Figure 9b that by increasing ω_2 , the population density of the infected class will reduce and the susceptible class will increase.

Finally, the impact of memory effect (α) is investigated. The numerical simulation is given by Figure 11. For $\alpha = 0.7, 0.8, 0.9, 1$ and similar initial values, all solution converge to single equilibrium point given by $\mathcal{E}_I \approx (1.3465, 1.0395)$, see Figure 11(a,b). We then plot the local amplification to show the difference of solutions when α is varied. We find that the difference

lies in the convergence rate where for larger values of α , the convergence rate increase and vice versa as shown in Figure 11(e,f). In the beginning, Figure 11(c,d) we show that when α decrease, the population density of the infected class reduce. From a biological point of view, we can say that biological memory has an impact on the density of both susceptible and infected classes.

6. CONCLUSION

The dynamics of a fractional-order SIS-epidemic model with intraspecific and interspecific competition have been studied. The validity of the model has been confirmed analytically by showing the existence, uniqueness, non-negativity, and boundedness of solutions. Three equilibrium points have been obtained namely the origin, the disease-free equilibrium point, and the endemic equilibrium point. Both origin and disease-free equilibrium points always exist while the endemic equilibrium point conditionally exists. The basic reproduction number \mathcal{R}_0 has been given which has a relationship with the local stability of the model. If $\mathcal{R}_0 < 1$ then the disease-free equilibrium point is locally asymptotically stable and if $\mathcal{R}_0 > 1$ then the disease-free equilibrium point losses its stability along with the existence of a locally asymptotically stable endemic equilibrium point. The global stability conditions of equilibrium points also have been found. The PRCC has been worked to investigate the most influential parameter. We have successfully shown that the infection rate and the death rate of the infected population due to interspecific competition becomes the most influential parameter for basic reproduction number and the population density of the infected class. We then investigate the impact of several parameters using numerical simulations including the infection rate, the recovery rate, the intraspecific competition, the interspecific competition, and the memory effect on the dynamics of the model. Bifurcation diagrams and time series have been given which show the existence of forward bifurcation, the decrease of susceptible and infected classes, and the decrease of convergence rate caused by the memory effect.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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