Qualitative analysis of a fractional-order two-strain epidemic model with vaccination and general non-monotonic incidence rate

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Received: 31 July 2022 / Revised: 7 October 2022 / Accepted: 10 November 2022 / Published online: 22 November 2022 © The Author(s), under exclusive licence to Springer-Verlag GmbH Germany, part of Springer Nature 2022

Abstract

In this paper, a fractional-order two-strain epidemic model with vaccination and general non-monotonic incidence rate is analyzed. The studied problem is formulated using susceptible, infectious and recovered compartmental model. A Caputo fractional operator is incorporated in each compartment to describe the memory effect related to an epidemic evolution. First, the global existence, positivity and boundedness of solutions of the proposed model are proved. The basic reproduction numbers associated with studied problem are calculated. Four steady states are given, namely the disease-free equilibrium, the strain 1 endemic equilibrium, the strain 2 endemic equilibrium, and the endemic equilibrium associated with both strains. By considering appropriate Lyapunov functions, the global stability of the equilibrium points is proven according to the model parameters. Our modeling approach using a generalized non-monotonic incidence functions encloses a variety of fractional-order epidemic models existing in the literature. Finally, the theoretical findings are illustrated using numerical simulations.

Keywords Caputo derivative · Two-strain · Vaccination · Non-monotonic incidence function · Global stability

1 Introduction and model formulation

Dynamic modeling using fractional differential equations has been a valuable tool for managing and controlling the behavior of many dynamical phenomena [1–5]. Due to the memory attribute of fractional derivative operator [1], fractional modeling gained significance importance to study complex phenomena with memory behavior, as is the case in many biological systems (see [2,6,11–13]). In this regard, several fractional-order models have studied epidemic dynamics through a system of fractional ordinary differential equations [6–10,14–20]. Many works involving fractional calculus are mainly focused on SIR-type models (see for instance [6,14,15,19]). Saeedian et al. [6] studied the effect of memory on the evolution of epidemics by means of fractional SIR epidemic model using the Caputo fractional operator. The authors in [15] studied and investigated the global stability of a fractional SIR epidemic model. Recently, Djilali et al. [20] presented and analyzed a fractional epidemic model with nonlinear incidence rate. Lately, Akdim et al. [19] have mainly studied the role of awareness campaigns strategies on the spread of an infectious disease using a Caputo fractional epidemic model with nonlinear-incidence function.

On the other hand, non-monotonic incidence rates are largely used in epidemic models in order to introduce the role of psychological effect in epidemics course [21–24]. Further, the COVID-19 pandemic crisis has highlighted the importance of psychological effect and vaccination strategy in controlling the spread of diseases [23,25]. We have also learned from this health crisis that during the evolution of a pandemic, several strains and variants of the virus can appear in the epidemic dynamics [26,27]. For this end, several works have dealt with vaccinated epidemic models [25,28–30], models with non-monotonic incidence rates [21,23,31,32], and multi-strain epidemic models [28,31,33]. Indeed, various methods can be used to model a vaccination strategy where vaccinated individuals can be assumed to be recovered population with permanent immunity (see [29]), or it can be approached by adding an additional com-
partment (as in [32]). However, it is well recognized that when public health authorities initiate a vaccination policy, it is always crucial to take the psychological effect and social awareness into account while launching a concurrent vaccination campaign. Obviously, the classical bilinear incidence rate does not take into account the force effect to control measures and social awareness when the number of infected becomes larger. For this reason, non-monotonic incidence rate functions have been widely investigated in the literature to describe these phenomena (see for instance [21,34,35]). In this paper, we consider a two-strain fractional-order SIR epidemic model with vaccination where an individual can be simultaneously infected with two strains of the same virus. Moreover, we consider the psychological effect during epidemic dynamics using a general non-monotonic incidence rate which encompasses many models with classical non-monotonic incidence functions (for instance \( \frac{I}{1+\alpha I^2} \) with \( \alpha > 0 \) [21] and \( \frac{I}{1+a_1 I + a_2 I^2} \) with \( a_1 > 0, a_2 > 0 \) [34]). Our problem is modeled using Caputo fractional dynamical system in order to capture the memory effects, history, or non-local effects that exist in many biological systems and exhibit the actual biphasic decline behavior of infection or illnesses but at a slower rate [6]. Therefore the proposed problem is modeled using the following fractional-order epidemic system

\[
\begin{align*}
D^\alpha S &= \Lambda - \beta_1 G_1(I_1)S - \beta_2 G_2(I_2)S - (\mu + v)S, \\
D^\alpha I_1 &= \beta_1 G_1(I_1)S - (\mu + d_1 + \gamma_1)I_1, \\
D^\alpha I_2 &= \beta_2 G_2(I_2)S - (\mu + d_2 + \gamma_2)I_2, \\
D^\alpha R &= vS + \gamma_1 I_1 + \gamma_2 I_2 - \mu R.
\end{align*}
\]

(1)

The studied population can be divided into four main compartments according to the epidemiological status of individuals: Susceptible population size (\( S(t) \)), strain 1 infectious individuals (\( I_1(t) \)), strain 2 infectious individuals (\( I_2(t) \)), and recovered (\( R(t) \)) population at time \( t \). The parameters of the above model are listed as follows, \( \Lambda \) is the recruitment rate, \( \mu \) is the natural death rate, while \( d_1 \) and \( d_2 \) are, respectively, the death rates due to strain 1 and strain 2 diseases, \( \gamma_1 \) and \( \gamma_2 \) are the recovery rates of the strain 1 and strain 2 infectious individuals, respectively. The general non-monotonic incidence function proposed by Cai et al. [36] is included for both strains in order to take into account the psychological effect during epidemic evolution in which

\[
G_i(I_i) = \frac{I_i}{f_i(I_i)}, \quad i = 1, 2.
\]

The component \( \frac{1}{f_i(I_i)} \) describes the force effect leading to the reduction of valid contact coefficient \( \beta \) (see [37]) such that

\[
0 < \frac{1}{f_i(I_i)} < \infty, \quad f_i'(I_i) > 0, \quad i = 1, 2
\]

and there is a \( m_i > 0 \) such that \( G_i(I_i)' > 0 \) for \( 0 < I_i \leq m_i \) and \( G_i(I_i)' < 0 \) for \( I_i \geq m_i \). If \( G_i(I_i) \) is a non-monotonic function, it means that \( G_i(I_i) \) is an increasing function when the infected population size is small and decreases when the number of infected individuals becomes large. Such mathematical property can be used to explain psychological effect during an epidemic: the infection force decreases when the size of infected individuals is increasing, which conduct to a reduction of the valid contacts per unit of time (for more details see [21,36,37]).

Since the three first ordinary equations are independent of the fourth equation, model (1) is limited to the following reduced problem:

\[
\begin{align*}
D^\alpha S &= \Lambda - \frac{\beta_1 I_1}{f_1(I_1)}S - \frac{\beta_2 I_2}{f_2(I_2)}S - (\mu + v)S, \\
D^\alpha I_1 &= \frac{\beta_1 I_1}{f_1(I_1)} - \delta_1 I_1, \\
D^\alpha I_2 &= \frac{\beta_2 I_2}{f_2(I_2)}S - \delta_2 I_2, \\
D^\alpha R &= vS + \frac{\gamma_1 I_1 + \gamma_2 I_2}{S(0)} - \mu R.
\end{align*}
\]

(2)

with \( \delta_1 = \mu + d_1 + \gamma_1 \) and \( \delta_2 = \mu + d_2 + \gamma_2 \).

The rest of this paper is outlined as follows. In the next section, we present some preliminary material and we show that the proposed problem is biologically and mathematically well posed. We calculate the corresponding basic reproduction numbers, and we investigate the existence of equilibria. Then we mainly study the global stability of the system according to the basic reproduction numbers. Finally, we resolve numerically the model and we present some simulations to validate our theoretical results.

2 Qualitative analysis

2.1 Preliminaries and global positive unique solution

Definition 1 The fractional integral of order \( \alpha > 0 \) of a function \( \varphi : \mathbb{R}_+ \to \mathbb{R} \) is defined as follows:

\[
I^\alpha \varphi(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - x)^{\alpha-1} \varphi(x)dx,
\]

where \( \Gamma(.) \) is the Gamma function.

Definition 2 The Caputo fractional derivative of order \( \alpha > 0 \) of a class \( C^n \) function \( \varphi : \mathbb{R}_+ \to \mathbb{R} \) is given by

\[
D^\alpha \varphi(t) = I^{n-\alpha} D^n \varphi(t),
\]

(4)
where $\alpha \in (n - 1, n)$, $n \in \mathbb{N}^*$. In particular, when $\alpha \in (0, 1)$, we have

\[
D_\alpha^\alpha \varphi(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{\varphi'(x)}{(t-x)^\alpha} dx. \tag{5}
\]

**Definition 3** (see [39]) The point $X^*$ is an equilibrium of the following Caputo fractional dynamical system

\[
D_\alpha^\alpha X(t) = h(t, X(t)), \quad \alpha \in (0, 1)
\]

if and only if, $h(t, X^*) = 0$.

**Lemma 1** (The generalized mean value theorem [40]) Suppose that $\varphi(x) \in C[a, b]$ and $D_\alpha^\alpha \varphi \in C(a, b]$, then

\[
\varphi(t) = \varphi(a) + \frac{1}{\Gamma(\alpha)} (D_\alpha^\alpha \varphi)(\zeta)(t-a)^\alpha,
\]

with $a \leq \zeta \leq t$, for all $t \in C \ (a, b]$. \hfill \Box

**Corollary 1** We consider that $\varphi(x) \in C[a, b]$ and $D_\alpha^\alpha \varphi \in C(\ [a, b]$, where $\alpha \in (0, 1)$. Then if

1. $D_\alpha^\alpha \varphi(x) \geq 0$, for all $x \in (a, b)$, then $\varphi(x)$ is non-decreasing.
2. $D_\alpha^\alpha \varphi(x) \leq 0$, for all $x \in (a, b)$, then $\varphi(x)$ is non-increasing.

Let $X(t) = \begin{pmatrix} S(t) \\ I_1(t) \\ I_2(t) \end{pmatrix}$, then we can reformulate system (3) as follows:

\[
D_\alpha^\alpha X(t) = H(X(t)),
\]

$X(0) \geq 0$. \hfill \Box

where

\[
H(X(t)) = \begin{pmatrix} \Lambda - \frac{\beta_1 I_1}{f_1(I_1)} S - \frac{\beta_2 I_2}{f_2(I_2)} S - (\mu + \nu) S \\ \frac{\beta_1 I_1}{f_1(I_1)} - \delta_1 I_1 \\ \frac{\beta_2 I_2}{f_2(I_2)} - \delta_2 I_2 \end{pmatrix}.
\]

We assume that all the functions $S, I_1, I_2, D^\alpha S, D^\alpha I_1$ and $D^\alpha I_2$ are continuous for all $t \geq 0$. To start the analysis of problem (3), we show the existence, uniqueness, and non-negativity of the solution. For this end we use the same argument as in [38]. Then we state the following theorem.

**Theorem 1** There exists a unique solution $X(t)$ of problem (3), which will remain in $\mathbb{R}^3_+$, $\{ X \in \mathbb{R}^3 \mid X \geq 0 \}$. Furthermore, the solution is non-negative.

**Proof** Applying Theorem 3.1. [41], we can easily prove the solution existence and by using remark 3.2 [41] we obtain the uniqueness of the solution for all $t > 0$. Let

\[
\varepsilon = \begin{pmatrix} \Lambda \\ 0 \\ 0 \end{pmatrix}, \quad A = \begin{pmatrix} -\mu - \nu & 0 & 0 \\ 0 & -\delta_1 & 0 \\ 0 & 0 & -\delta_2 \end{pmatrix},
\]

\[
B = \begin{pmatrix} -\beta_1 & -\beta_2 \\ \beta_1 & \beta_2 \end{pmatrix}, \quad C = \begin{pmatrix} I_1 S \\ f_1(I_1) \\ f_2(I_2) \end{pmatrix}
\]

then $H(X)$ can be written as follows:

\[
H(X) = \varepsilon + AX + BC.
\]

Then

\[
\| H(X) \| \leq \| \varepsilon \| + \| B \| \| C \| + \| A \| \| X \|.
\]

It follows by Theorem 3.1, [41] that the system has a unique solution. Next, we prove the non-negativity of the solution. From model (3) we have

\[
D^\alpha S(S=0) = \Lambda \geq 0,
\]

\[
D^\alpha I_1(S=0) = 0 \geq 0,
\]

\[
D^\alpha I_2(S=0) = 0 \geq 0.
\]

Therefore, by using Corollary 1, we obtain the desired result, i.e., the solution will remain in $\mathbb{R}^3_+$. \hfill \Box

From above theorem we concluded that the feasible region of model (3) is as follows:

\[
\Delta = \left\{ (S, I_1, I_2) \in \mathbb{R}^3_+ : S, I_1, I_2 \geq 0 \right\}.
\]

### 2.2 The basic reproduction number and equilibria

In epidemiology, the basic reproduction number $R_0$ [42] is defined as the expected average number of new infected cases caused by one case in a susceptible population. It can be determined theoretically using the next generation matrix. We calculate the basic reproduction number as a spectral radius of $F V^{-1}$, where $F$ is the non-negative matrix of new infection cases, and $V$ is the the transition infections matrix associated with the studied epidemic model.

\[
F = \begin{pmatrix} \frac{\beta_1 \Lambda}{(\mu + \nu) f_1(0)} \\ 0 \\ \frac{\beta_2 \Lambda}{(\mu + \nu) f_2(0)} \end{pmatrix}, \quad V = \begin{pmatrix} \delta_1 \\ 0 \\ \delta_2 \end{pmatrix}
\]
So
\[ F^{\nu - 1} = \left( \frac{\beta_1 A}{(\mu + v) f_1(0) \delta_1} 0 \frac{\beta_2 A}{(\mu + v) f_2(0) \delta_2} \right). \]

Hence
\[ R_0 = \max \{ R_0^1, R_0^2 \}, \]
with
\[ R_0^1 = \frac{\beta_1 A}{(\mu + v) f_1(0) \delta_1}, \]
\[ R_0^2 = \frac{\beta_2 A}{(\mu + v) f_2(0) \delta_2}. \]

\( f_1(0) \) and \( f_2(0) \) are positive constants according to condition (2).

Now, we discuss the steady states of the model. Then we can see that the model has disease-free equilibrium and three endemic equilibria as follows:

- The disease-free equilibrium \( E^0 = (\frac{\Lambda}{\mu + v}, 0, 0) \).
- The strain 1 endemic equilibrium \( E^{*1} = (S^{*1}, I_1^{*1}, I_2^{*1}) \), where
  \[ S^{*1} = \frac{\delta_1 f_1(I_1^{*1})}{\beta_1}, \quad I_1^{*1} = 0 \]
  and \( I_1^{*1} \) is determined by
  \[ \Lambda - \delta_1 I_1^{*1} f_1(I_1^{*1}) - (\mu + v) \frac{\delta_1 f_1(I_1^{*1})}{\beta_1} = 0. \]
- The strain 2 endemic equilibrium \( E^{*2} = (S^{*2}, I_1^{*2}, I_2^{*2}) \), where
  \[ S^{*2} = \frac{\delta_2 f_2(I_2^{*2})}{\beta_2}, \quad I_1^{*2} = 0 \]
  and \( I_2^{*2} \) is determined by
  \[ \Lambda - \delta_2 I_2^{*2} f_2(I_2^{*2}) - (\mu + v) \frac{\delta_2 f_2(I_2^{*2})}{\beta_2} = 0. \]
- The endemic equilibrium \( E^{*} = (S^{*}, I_1^{*}, I_2^{*}) \), where
  \[ S^{*} = \frac{\beta_1 I_1^{*} + \beta_2 I_2^{*}}{f_1(I_1^{*}) + f_2(I_2^{*})} + \frac{\nu + \mu}{\beta_1} \]
  in which \( I_1^{*} \) and \( I_2^{*} \) are determined by
  \[ \Lambda - \delta_1 I_1^{*} - (\mu + v) \frac{\delta_1 f_1(I_1^{*})}{\beta_1} = 0, \]
  \[ \Lambda - \delta_1 I_1^{*} - (\mu + v) \frac{\delta_2 f_2(I_2^{*})}{\beta_2} = 0. \]

**Theorem 2** Problem (3) has the disease-free equilibrium \( E^0 \) and three endemic equilibria \( E^{*1}, E^{*2} \) and \( E^{*} \). Moreover, we have

- The strain 1 endemic equilibrium \( E^{*1} \) exists when \( R_0^1 > 1 \).
- The strain 2 endemic equilibrium \( E^{*2} \) exists when \( R_0^2 > 1 \).
- The third endemic equilibrium \( E^{*} \) exists when \( R_0^1 > 1 \) and \( R_0^2 > 1 \).

**Proof** The equilibria of model (3) are obtained by solving the system
\[ D^a S = D^a I_1 = D^a I_2 = 0. \]

We consider the functions \( h^k, k = 1, 2 \) defined as
\[ h^k(x) = \Lambda - \delta_k f_k(x) - (\mu + v) \frac{\delta_k f_k(x)}{\beta_k}, \quad k = 1, 2. \]

Then we prove that the equation \( h^k(x) = 0 \) admits a unique solution. We have
\[ h^k(x) = -\delta_k f_k'(x) - (\mu + v) \frac{\delta_k f_k(x)}{\beta_k}. \]

Obviously, we can see that \( h^k(x) < 0 \); then, \( h^k(x) \) is decreasing functions. On the other hand, since \( f_k \) are positive increasing functions, we have \( \lim_{x \to \infty} f_k(x) = \infty \) and
\[ h^k(0) = \Lambda - (\mu + v) \frac{\delta_k f_k(0)}{\beta_k} = \Lambda \left( 1 - \frac{1}{R_0^k} \right), \quad k = 1, 2. \]

If \( R_0^k > 1 \) then \( h^k(0) > 0 \). Therefore, according to the intermediate value theorem and since \( h^k(x) \) are decreasing functions, there is a unique solution of the equation \( h^k(x) = 0 \). Hence, if \( R_0^1 > 1 \) the system has a unique strain 1 endemic equilibrium \( E^{*1} \) and a unique strain 2 endemic equilibrium \( E^{*2} \) when \( R_0^2 > 1 \).

Let
\[ F^1(x) = \Lambda - \delta_1 x - (\mu + v) \frac{\delta_1 f_1(x)}{\beta_1}. \]
In this section, we investigate the global stability of the disease-free equilibrium $E^0$ and the endemic equilibrium $E^\ast$.

Theorem 3 If $R_0 \leq 1$, the disease-free equilibrium $E^0$ is globally asymptotically stable.

Proof We consider the following Lyapunov function:

$$\Psi_0(t) = \left( S - S_0 - S_0 \log \frac{S}{S_0} \right) + I_1 + I_2,$$

with $S_0 = \frac{\Lambda}{\mu + \nu}$. Then we calculate the fractional time derivation of $\Psi_0$ along the solution of system (3). We get

$$D^\alpha \Psi_0(t) \leq \left(1 - \frac{S_0}{S}\right) D^\alpha S + D^\alpha I_1 + D^\alpha I_2,$$

using the fact that $\Lambda = (\mu + \nu) S_0$, we obtain

$$D^\alpha \Psi_0(t) \leq -\frac{\mu + \nu}{S} (S - S_0)^2 - \frac{\beta_1(S - S_0)}{f_1(I_1)} I_1 - \frac{\beta_1(S - S_0)}{f_2(I_2)} I_2 + \frac{S_0 I_1}{f_1(I_1)} + \frac{S_0 I_2}{f_2(I_2)} - \delta_1 I_1 - \delta_2 I_2.$$

Using the fact that $f_i(I_i), i = 1, 2$ are increasing functions, then $\frac{1}{f_i(I_i)} \leq \frac{1}{f_i(0)}, i = 1, 2$. It follows that

$$D^\alpha \Psi_0(t) \leq -\frac{\mu + \nu}{S} (S - S_0)^2 + \frac{S_0 I_1}{f_1(0)} + \frac{S_0 I_2}{f_2(0)} - \delta_1 I_1 - \delta_2 I_2.$$

Since $R_0 \leq 1$, i.e., $R_0^1 \leq 1$ and $R_0^2 \leq 1$, then $D^\alpha \Psi_0(t) \leq 0$. Furthermore $D^\alpha \Psi_0(t) = 0$ holds, if and only if $S = S_0$ and $I_1 = 0, I_2 = 0$. Consequently, the largest invariant set of $(S, I_1, I_2) \in \mathbb{R}_+^3 : D^\alpha \Psi_0(t) = 0$ is the singleton $\{E^0\}$. According to the fractional LaSalle’s invariance principle [2], $E^0$ is globally asymptotically stable.

Theorem 4 The endemic equilibrium $E^\ast$ is globally asymptotically stable whenever $R_0^1 > 1$ and $R_0^2 > 1$.

Proof We consider the following Lyapunov function:

$$\Psi^\ast(t) = \frac{1}{2} (S - S^\ast + I_1 + I_2 - I_1^\ast - I_2^\ast)^2 + c_1 \left( I_1 - I_1^\ast - \log \frac{I_1}{I_1^\ast} \right) + c_2 \left( I_2 - I_2^\ast - \log \frac{I_2}{I_2^\ast} \right),$$

where $c_1$ and $c_2$ are positive constants to be determined later. We calculate the fractional time derivation of $\Psi^\ast$ along the solution of system (3); we get

$$D^\alpha \Psi^\ast(t) \leq (D^\alpha S + D^\alpha I_1 + D^\alpha I_2) (S - S^\ast + I_1 - I_1^\ast + I_2 - I_2^\ast) + c_1 \left( I_1 - I_1^\ast \right) \left( \frac{S_1}{f_1(I_1^\ast)} - \delta_1 I_1 \right) + c_2 \left( I_2 - I_2^\ast \right) \left( \frac{S_1}{f_2(I_2^\ast)} - \delta_2 I_2 \right).$$

Hence

$$D^\alpha \Psi^\ast(t) \leq (S - S^\ast + I_1 - I_1^\ast + I_2 - I_2^\ast) \left( \frac{\mu + \nu}{S} (S - S_0)^2 + \frac{S_0 I_1}{f_1(I_1)} + \frac{S_0 I_2}{f_2(I_2)} - \delta_1 I_1 - \delta_2 I_2 \right).$$

Using the fact that

$$\delta_1 = \frac{\beta_1 S^\ast}{f_1(I_1^\ast)} \text{ and } \delta_2 = \frac{\beta_2 S^\ast}{f_2(I_2^\ast)},$$

we obtain

$$D^\alpha \Psi^\ast(t) \leq (S - S^\ast + I_1 - I_1^\ast + I_2 - I_2^\ast) \left( \left( \frac{\mu + \nu}{S} (S - S_0)^2 + \frac{S_0 I_1}{f_1(I_1)} + \frac{S_0 I_2}{f_2(I_2)} - \delta_1 I_1 - \delta_2 I_2 \right) \left( \frac{1}{f_1(I_1^\ast)} - 1 \right) S \right) + \frac{c_1 \beta_1}{f_1(I_1^\ast)} (I_1 - I_1^\ast) (S - S^\ast) - c_1 \beta_1 (I_1 - I_1^\ast) \left( \frac{1}{f_1(I_1^\ast)} - 1 \right) S$$

$$+ \frac{c_2 \beta_2}{f_2(I_2^\ast)} (I_2 - I_2^\ast) (S - S^\ast) - c_2 \beta_2 (I_2 - I_2^\ast) \left( \frac{1}{f_2(I_2^\ast)} - 1 \right) S.$$

Since $R_0^1 > 1$, i.e., $R_0^1 \leq 1$ and $R_0^2 \leq 1$, then $D^\alpha \Psi^\ast(t) \leq 0$.
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Fig. 1 Global behavior of susceptible population as function of time for $\Lambda = 7$, $\mu = 0.08$, $\nu = 0.01$; $\beta_1 = 0.0015$; $\beta_2 = 0.006$; $d_1 = 0.1$; $d_2 = 0.1$; $\gamma_1 = 0.4$; $\gamma_2 = 0.3$; $k_1 = 0.1$; $k_2 = 0.1$ and different initial conditions $S(0) = 1000; 600; 300$ which corresponds to the stability of the disease-free equilibrium $E^0$.

Choose $c_i = \frac{\mu + \delta_i}{\beta_i} f_i(I^*_i)$, $i = 1, 2$, since $f_i(x)$ are increasing functions we have $(I_i - I^*_i) \left( \frac{1}{f_i(I^*_i)} - \frac{1}{f_i(I_i)} \right) \geq 0$ for $i = 1, 2$. Then we get

$$D^\alpha \Psi^*(t) \leq - (\mu + \nu) (S - S^*)^2 - \delta_1 (I_1 - I^*_1)^2 - \delta_2 (I_2 - I^*_2)^2$$

Fig. 2 Global behavior of strain 1 infection as function of time for $\Lambda = 7$, $\mu = 0.08$, $\nu = 0.01$; $\beta_1 = 0.0015$; $\beta_2 = 0.006$; $d_1 = 0.1$; $d_2 = 0.1$; $\gamma_1 = 0.4$; $\gamma_2 = 0.3$; $k_1 = 0.1$; $k_2 = 0.1$ and different initial conditions $I_1(0) = 100; 20; 5$ which corresponds to the stability of the disease-free equilibrium $E^0$.
Therefore, \( D^\omega \Psi^* (t) \leq 0 \). Hence, the largest invariant set of \( \{(S, I_1, I_2) \in \mathbb{R}^3_+ \colon D^\omega \Psi^*(t) = 0\} \) is the singleton \( \mathcal{E}^* \). By fractional LaSalle’s invariance principle [2], \( \mathcal{E}^* \) is globally asymptotically stable.

Choosing the functions

\[
\psi^{*1} (t) = \frac{1}{2} (S - S^* + I_1 - I_1^*)^2 + c_1 \left( I_1 - I_1^* - l_1^* \log \frac{I_1^*}{I_1} \right)
\]

\[
\psi^{*2} (t) = \frac{1}{2} (S - S^* + I_2 - I_2^*)^2 + c_2 \left( I_2 - I_2^* - l_2^* \log \frac{I_2^*}{I_2} \right).
\]

Then from (20) we deduce

\[
D^\omega \psi^{*1} (t) \leq - (\mu + v) (S - S^*)^2 - \delta_1 (I_1 - I_1^*)^2 - c_1 \beta_1 (I_1 - I_1^*) \left( \frac{1}{f_1 (I_1^*)} - \frac{1}{f_1 (I_1)} \right) S.
\]

(21)

\[
D^\omega \psi^{*2} (t) \leq - (\mu + v) (S - S^*)^2 - \delta_2 (I_2 - I_2^*)^2 - c_2 \beta_2 (I_2 - I_2^*) \left( \frac{1}{f_2 (I_2^*)} - \frac{1}{f_2 (I_2)} \right) S.
\]

(22)

Obviously, from the above inequalities we can obtain the following result:

**Corollary 2**  
- The strain 1 endemic equilibrium \( \mathcal{E}^{*1} \) is globally asymptotically stable whenever \( R_0^2 < 1 < R_0^1 \).
- The strain 2 endemic equilibrium \( \mathcal{E}^{*2} \) is globally asymptotically stable whenever \( R_0^1 < 1 < R_0^2 \).

### 3 Numerical investigations

In this section, we give some numerical simulations to illustrate our analytical results. For this end, we consider the non-monotonic incidence function presented by Xiao and Ruan in [21] as follows:

\[
G_1(I_1) = \frac{I_1}{f_1(I_1)} = I_1 I_1^*,
\]

\[
G_2(I_2) = \frac{I_2}{f_2(I_2)} = I_2 I_2^*.
\]

Then the fractional-order epidemic system becomes:

\[
\begin{align*}
D^\omega S(t) &= \Lambda - \beta_1 I_1(t) S(t) - \beta_2 I_2(t) S(t) - (\mu + v) S(t), \\
D^\omega I_1(t) &= \beta_1 \frac{I_1(t) S(t)}{1 + k_1 I_1(t)} - \delta_1 I_1(t), \\
D^\omega I_2(t) &= \beta_2 \frac{I_2(t) S(t)}{1 + k_2 I_2(t)} - \delta_2 I_2(t),
\end{align*}
\]

(23)

It is easy to see that

\[
R_0 = \max \{ R_0^1, R_0^2 \} = \max \left\{ \frac{\beta_1 \Lambda}{(\mu + v) \delta_1}, \frac{\beta_2 \Lambda}{(\mu + v) \delta_2} \right\}.
\]

We apply the numerical scheme presented by Erturk et al. [43] to solve problem (23). We consider the following equivalent problem

\[
\begin{align*}
D^\omega X(t) &= H (X(t)), \\
X(0) &= X_0, \quad X_0 \in \mathbb{R}^3_+
\end{align*}
\]

(24)

with \( X_0 = \left( \begin{array}{c} S(0) \\ I_1(0) \\ I_2(0) \end{array} \right) \). Then the problem is resolved using the following numerical scheme

\[
H (t_j) = \frac{k^\alpha}{\Gamma(\alpha + 2)} \left( (j - 1)^{\alpha + 1} - (j - \alpha - 1)^{\alpha + 1} \right)
\]

\[
+ \frac{k^\alpha}{\Gamma(\alpha + 2)} \sum_{i=1}^{j-1} \left( (j - i + 1)^{\alpha + 1} - (j - i)^{\alpha + 1} \right)
\]

\[
H (X (t_j)) + X (0)
\]

\[
+ \frac{k^\alpha}{\Gamma(\alpha + 2)} H \left( X (t_{j-1}) + \frac{k^\alpha}{\Gamma(\alpha + 1)} H \left( X (t_{j-1}) \right) \right)
\]

(25)

with \( t_{j+1} = t_j + h \), for \( j = 0, 1, \ldots, N - 1 \), we used \( (N = 5000) \).

Figures 1, 2 and 3 depict the behavior of the infection in observation time. The basic reproduction number \( R_0 \) is less than unity \( (R_0 = 0.97 < 1, R_0^1 = 0.2 < 1, R_0^2 = 0.97) \). It is shown that the curves converge toward the disease-free-equilibrium \( \mathcal{E}^0 = (77, 0, 0) \) even if the initial conditions are different which confirm the global stability result of \( \mathcal{E}^0 \).

The stability behavior of the endemic steady-state \( \mathcal{E}^* \) is illustrated in Figs. 4, 5 and 3. In this case, the basic reproduction numbers exceed the unity \( (R_0^1 = 3.25 > 1, R_0^2 = 5.9 > 1) \). It was observed that for different initial conditions the curves converge to the endemic equilibrium point \( \mathcal{E}^* = (81, 4, 15) \) which confirm the global stability result of \( \mathcal{E}^* \).
Figure 3 Global behavior of strain 2 infection as function of time for $\Lambda = 7$, $\mu = 0.08$, $v = 0.01; \beta_1 = 0.0015; \beta_2 = 0.006; d_1 = 0.1; d_2 = 0.1; \gamma_1 = 0.4; \gamma_2 = 0.3; k_1 = 0.1; k_2 = 0.1$ and different initial conditions $I_2(0) = 20; 8; 2$ which corresponds to the stability of the disease-free equilibrium $E^0$.

Figure 4 Global behavior of susceptible population as function of time for $\Lambda = 17$, $\mu = 0.08$, $v = 0.01; \beta_1 = 0.01; \beta_2 = 0.015; d_1 = 0.1; d_2 = 0.1; \gamma_1 = 0.4; \gamma_2 = 0.3; k_1 = 0.1; k_2 = 0.1$ and different initial conditions $S(0) = 1000; 600; 300$ which corresponds to the stability of the endemic equilibrium $E^*$.

Figure 7 shows the dynamical behavior of the infection when $R_0^1 = 0.83 < 1$ and $R_0^2 = 4.43$. It is observed that the trajectories converge toward the strain 2 endemic equilibrium $E^*$. Even if the initial conditions are different which confirm the global stability result of $E^*$ according to above Corollary 2. Further, the global dynamical stability behavior of the strain 1 infection is illustrated in Fig. 8 where $R_0^1 = 5.9 > 1$ and $R_0^2 = 0.59 < 1$ for different initial conditions. We observe that the trajectories converge toward the strain 1 endemic state $E^*$. Have confirmed the result in Corollary 2.

Figure 9 depicts the dynamics behavior of both strains infection illustrating how the vaccine parameter can affect the evolution of an epidemic when the disease is persistent ($R_0^1 > 1$ and $R_0^2 > 1$) over time. Then, we can observe that increasing the values of vaccine rate $v = 0.001; 0.1; 0.3$...
leads to a decrease in basic reproduction numbers $R^1_0$, $R^2_0$ and then a reduction in infectious individuals.

The previous examples show that the stability of the equilibria is unaffected by the order of the fractional derivative. However, the solutions converge to steady states more quickly for higher values of $\alpha$, which represent the long memory term; this can support the idea that epidemic dynamics is directly tied to the people’s experiences, social awareness, memories, and knowledge induced from epidemics [6]. Furthermore, it can be seen that the response of the fractional-order model gives more trajectories by changing the fractional parameter $\alpha$; this advantage can improve real data fitting. Indeed, when fitting data, the fractional models have one more degree of freedom than the integer-order model.
Fig. 7 Dynamics behavior of both strains infection (strain 1 (left), strain 2 (right)) as function of time for $\Lambda = 17$, $\mu = 0.08$, $\nu = 0.04$; $\beta_1 = 0.004$; $\beta_2 = 0.015$; $d_1 = 0.1$; $d_2 = 0.1$; $\gamma_1 = 0.5$; $\gamma_2 = 0.3$; $k_1 = 0.4$; $k_2 = 0.1$ and different initial conditions $I_1(0) = 100; 20; 5$, $I_2(0) = 20; 8$; and $S(0) = 1000; 600; 300$ which corresponds to the stability of the strain 2 endemic equilibrium $E^2_1$.

Fig. 8 Dynamics behavior of both strains infection (strain 1 (left), strain 2 (right)) as function of time for $\Lambda = 17$, $\mu = 0.08$, $\nu = 0.01$; $\beta_1 = 0.015$; $\beta_2 = 0.0015$; $d_1 = 0.1$; $d_2 = 0.1$; $\gamma_1 = 0.3$; $\gamma_2 = 0.3$; $k_1 = 0.1$; $k_2 = 0.4$ and different initial conditions $I_1(0) = 100; 20; 5$, $I_2(0) = 20; 8$; and $S(0) = 1000; 600; 300$ which corresponds to the stability of the strain 1 endemic equilibrium $E^1_1$.

4 Conclusion

In this paper, we have proposed and analyzed a fractional-order two-strain epidemic model with vaccination and a general non-monotonic incidence. We have proved that studied model (3) is mathematically and biologically well posed as this is indispensable in population dynamics. Then we have calculated the basic reproduction numbers corresponding to the model, namely the strain 1 reproduction number $R^1_0$, the strain 2 reproduction number $R^2_0$ and the main basic reproduction number $R_0 = \max \{ R^1_0, R^2_0 \}$. Four steady states are found, that is the disease-free equilibrium point, the strain 1 endemic equilibrium point with respect to strain 1 infection, the strain 2 endemic equilibrium and the endemic equilibrium which correspond to the existence of both strains infection. Next, by using appropriate Lyapunov functions and according to the basic reproductions numbers thresholds, we have proved the global stability of all given equilibrium points. Finally, some numerical simulations examples are given so that our theoretical findings are supported. We have illustrated the effect of different vaccination rates on the size of both strains infectious population. It was observed that it was shown that changing the values of the fractional-order parameter $\alpha$ affects how long the variables take to reach the equilibrium points, but have no impact on the stability of equilibria. Further, it is shown that increasing the value of vaccination parameter decreases the magnitude of the infection. Our research may help to clarify the function of various...
control measures, such as immunization by vaccination in order to stop the illness from spreading widely among the population.

For further research papers, it will be interesting to study a more accurate problems, for instance using a fractional-order multi-strain epidemic models to investigate the epidemic dynamics in the presence of various variants of the same virus. Also, it will be interesting to study model (3) with time delay (see, for instance, [44]) and with more realistic environment by introducing fractional noise [45] or stochastic Lévy noise (see, e.g., [29,46,47]).

**Author Contributions** All authors contributed equally to the writing of this paper. They read and approved the final version of the manuscript.

**Funding** Not Applicable

**Availability of data and material** Not applicable.

**Code Availability** Available on request from the corresponding author.

**Declarations**

Conflict of interest The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in the above mentioned paper.

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