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On nonlinear dynamics of a fractional order monkeypox virus model

A. El-Mesady\textsuperscript{a}, Amr Elsonbaty\textsuperscript{b,c}, Waleed Adel\textsuperscript{c,d,*}

\textsuperscript{a} Department of Physics and Engineering Mathematics, Faculty of Electronic Engineering, Menoufiya University, Menouf 32952, Egypt
\textsuperscript{b} Department of Mathematics, College of Science and Humanities in Al-Kharj, Prince Sattam bin Abdulaziz University, Al-Kharj 11942, Saudi Arabia
\textsuperscript{c} Department of Mathematics and Engineering Physics, Faculty of Engineering, Mansoura University, Mansoura, Egypt
\textsuperscript{d} Université Française d’Egypte, Lomailia Desert Road, El-Shorouk, Cairo, Egypt

\textbf{Abstract}

In this work, we examine a fractional-order model for simulating the spread of the monkeypox virus in the human host and rodent populations. The employment of the fractional form of the model gives a better insight into the dynamics and spread of the virus, which will help in providing some new control measures. The model is formulated into eight mutually exclusive compartments and the form of a nonlinear system of differential equations. The reproduction number for the present epidemic system is found. In addition, the equilibrium points of the model are investigated and the associated stability analysis is carried out. The influences of key parameters in the model and the ways to control the monkeypox epidemic have been thoroughly examined for the fractional model. To ensure that the model accurately simulates the nonlinear phenomenon, we adapt an efficient numerical technique to solve the presented model, and the acquired results reveal the dynamic behaviors of the model. It is observed that when memory influences are considered for the present model, through Caputo fractional-order derivatives, they affect the speed and time taken by solution trajectories towards steady-state equilibria.

\textbf{1. Introduction}

During the last two years, researchers have given an increasing considerable amount of attention to viruses, trying to find treatment for such diseases. Since the pandemic at the end of 2019 and up until now, the need to find such a cure has been a must, and with the new variants that are appearing, there has been extensive work in this field. Monkeypox is one of the new viruses that has been re-appearing since the beginning of June 2022 and has drawn a large amount of attention from researchers trying to find some new prevention tools to control such viruses. From May 13 to May 21, 2022, 92 confirmed cases around the world have been reported in several non-endemic countries with no deaths until now \cite{1}. This is not the first time there has been an outbreak of monkeypox virus. This virus was first identified back in 1970 in Congo, a region that had been infected with smallpox. Since then, most of the reported cases have been reported to be in some rural areas in central and western Africa. Since this breakdown in Congo, other areas, including Benin, Cameroon, Gabon, Nigeria, and others, have been infected with the virus. During the year 2017, Nigeria was one of the most affected places by this virus with a total of 500 suspected cases, 200 confirmed cases, and a fatality rate of 3 %, and the cases are continuing to be reported until today. Another outbreak outside Africa occurred in 2007 in the United States of America and was directly linked to an infected pet to be considered patient zero. This outbreak led to 70 confirmed cases. With the ongoing discovery of confirmed cases, further studies need to be developed to understand the epidemiology, transmission patterns, and source of infection of monkeypox and also develop some treatments and vaccines.

Monkeypox is a viral zoonotic disease with moderate to severe symptoms that are thought to be transmitted from animal to human through direct contact with animals in rural and urban areas of rainforests in central and southern Africa. The monkeypox virus belongs to the family of Orthopox viruses similar to variola viruses, which is mainly the origin of the smallpox virus, vaccinia virus, and cowpox virus \cite{2,3}. Since this family of viruses is transmitted through animals, the hosts for this virus include various types of animals, such as rodents, prairie dogs, squirrels, and chimpanzees. With these types of hosts, monkeypox can be considered impossible to eliminate since the main source of transmission may develop to be from human to human. The main infection strategy is through the direct contact of the infected patient's fluids or respiratory droplets through the contaminated person's environment or items \cite{1,4}. With this type of infectious transmission, the most affected

\textsuperscript{*} Corresponding author at: Department of Mathematics and Engineering Physics, Faculty of Engineering, Mansoura University, Mansoura, Egypt.

E-mail addresses: AHMED.IBRAHIEM81@el-eng.menofia.edu.eg (A. El-Mesady), waleedadel85@yahoo.com (W. Adel).

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people are healthcare workers, since this type of infection requires close contact with the infected person, which exposes them to a high risk. In recent years, the most well-known chain of infection transmission from person to person has grown from 6 to 9 successive in-person transmissions. Another form of transmission of infection is through the placenta of the infected mother to her fetus during labor.

The most common symptoms of monkeypox are headache, fever, muscle pain, chills, fatigue, and swollen lymph nodes. The severity of the monkeypox virus may vary from person to person, with a death rate of 10%, mostly in children at the age of 10 years old [5]. The incubation period of a monkeypox virus-infected person can last from a week to two weeks, and it can last up to three weeks depending on the amount of infection the person attracts [6]. In the first few days, ranging from the first day of infection, some skin lesions may appear, growing into multiple small fluid-filled blisters with a bus filled. These diverse symptoms urge scientists to try to find some treatments and cures for this virus to stop its spread and take some control prevention measures.

Nowadays, the treatment of the monkeypox virus is not known, and the use of several antivirals, including Brincidofovir and Tecovirimat vaccines, for possible treatment and prevention of the infection. With no available effective vaccine, the number of infected cases has been increasing over the last few years, and recently in 2022, during a small outbreak in some small countries, urged WHO to look up the reasons and control prevention. The increased number of infections was associated with a decrease in herd immunity to the smallpox virus. The available vaccines have proven to be able to prevent the infection with a success rate of 85%, but they are no longer available due to the global eradication of smallpox. Vaccinated individuals who previously got the vaccine have less severe symptoms compared with nonvaccinated individuals, proving the effectiveness of the vaccine in stopping the spread of the virus [7,8].

The spread of the monkeypox virus has received little attention in the past, with few works studying the dynamics and spread of the virus. With the appearance of new cases in 2022, researchers began to delve deeper into the new spread to learn more about the transmission's behavior. For example, Bhaunu et al. [9] investigated the spread of the disease and concluded that with control intervention and treatment, the disease would vanish among those infected by it. Usman et al. [10] study the dynamics of monkeypox infection in both human and rodent populations along with the stability analysis of the presented model. Peter et al. [11] introduced a deterministic mathematical model that takes into account different values of control intervention and also studies the local and global stability of the proposed system. The outbreak of the monkeypox virus in 2022 has been studied by Haider et al. [12], giving some explanation of the reason for this emerging new case. They proved that the increase in the interaction of the rodent with humans led to an increased transmission rate for a human-to-human infection, also estimating the threshold number. In addition, the re-emergence of the monkeypox virus has been reconsidered by Shailendra et al. [13] as a global health crisis and the need for fast intervention actions to stop its spread and learn more about its transmission pattern and new treatment techniques. For more information, the reader may refer to [14–18] and the references therein.

Fractional calculus is one of the most important branches of mathematics that has extensive use while simulating epidemics, especially the ones with their symptoms, which take some time to appear. These viruses may include the COVID-19 virus, which may take up to 14 days for symptoms to appear, much like the monkeypox virus. It is known that there is an immediate connection between the time evolution of infectious diseases, including COVID-19, monkeypox, and the immunological memory of immune systems. In other words, the immune system in the human body can recognize previously encountered antigens and hence quickly initiate the associated immune response. Fractional order differential equations employ nonlocal operators, which are ideal tools in the mathematical modeling of systems with memory. For the previously mentioned reasons, the use of fractional calculus with different definitions has captured the interest and attention of researchers over the past few years, with its different properties to be applied in the field of disease simulations. Many researchers have been trying to use the different forms and definitions of the fractional order in their simulations, resulting in a better understanding of the disease dynamics. These definitions may include Caputo fractional operator [19,20], Grunwald-Letnikov [21], conformable fractional operator [22], and several other definitions. Each of these definitions has its advantages and disadvantages while simulating different forms of complex real-life phenomena. For example, Iziad et al. [23] applied a computational approach for simulating the fractional Lotka–Volterra population prey-predator model, revealing some of its new dynamics. Singh et al. [24] applied a computational approach based on the Legendre scaling function to simulate different cases of the fractional vibration equation. In addition, the fractional advection-dispersion equation that has some application in porous medium simulation has been solved using the Jacobi collocation approach by Singh et al. [25]. Elsonbaty et al. [26] investigated the dynamics of a fractional-order SITRS model that simulates the spread of the COVID-19 pandemic, taking into account the loss of temporary immunity. Also, the COVID-19 disease model has been studied in [27], giving new insight into the transmission behavior of the virus. The numerical simulation and stability analysis of a fractional COVID-19 model are being examined with the aid of an efficient technique presented by Singh et al. [28], revealing some new results for long-term behaviors. Recent studies on the use of fractional models simulating diseases are Ebola virus [29,30], childhood disease models [31], SEAIR epidemic model with optimal control [32], dengue epidemic model [33], measles epidemic model [34], SVEIR epidemic model [35], computer virus epidemic model [36], Hepatitis B model [37], dual variants of COVID-19 and HIV co-infection model [38], fractional order smoking model [39], fractional HIV infection model of CD4+ T-cells [40], and Lassa hemorrhagic fever disease [41].

Our primary focus in this paper is to investigate the effects of various factors in the mathematical modeling that simulates the spread of the disease. It is worth mentioning that this is the first time a fractional order model has been presented to simulate the monkeypox virus since its breakdown. The novelty of the proposed research can be pointed out in the next few points:

1. A newly designed fractional model defined in the Caputo sense for investigating the new dynamics of the monkeypox virus is examined.
2. The reproduction number $R_0$ for the proposed model is being derived along with both the state-free and endemic equilibrium points for the system.
3. The stability region for various values of the parameters is plotted.
4. An efficient numerical technique is adapted to verify the theoretical aspects and findings of the proposed model.
5. These acquired results prove that the proposed model is efficient in giving some new ideas about the dynamical behavior of such disease and providing some control measures.
human and rodent populations. The human population is further classified into five categories, including susceptible humans $S_h(t)$, exposed humans $E_h(t)$, infected humans $I_h(t)$, isolated humans $Q_h(t)$, and recovered humans $R_h(t)$. The rodent population is classified into three categories, including susceptible rodents $S_r(t)$, exposed rodents $E_r(t)$, and infected rodents $I_r(t)$. The main model can be formulated as the following nonlinear system \[11\].

\[
\begin{align*}
\frac{dS_h}{dt} &= \theta_h - \left(\frac{\beta_1 I_h + \beta_2 I_r}{N_h}\right) S_h - \mu_h S_h + \psi Q_h \\
\frac{dE_h}{dt} &= \left(\frac{\beta_1 I_h + \beta_2 I_r}{N_h}\right) S_h - (\alpha_1 + \alpha_2 + \mu_h) E_h \\
\frac{dI_h}{dt} &= \alpha_2 E_h - (\mu_h + \delta_h + \gamma) I_h \\
\frac{dQ_h}{dt} &= \alpha_2 E_h - (\varphi + \tau + \delta_h + \mu_h) Q_h \\
\frac{dR_h}{dt} &= \gamma I_h + \tau Q_h - \mu_h R_h \\
\frac{dS_r}{dt} &= \theta_r - \left(\frac{\beta_1 I_h + \beta_2 I_r}{N_r}\right) S_r - \mu_r S_r \\
\frac{dE_r}{dt} &= \left(\frac{\beta_1 I_h + \beta_2 I_r}{N_r}\right) S_r - (\alpha_1 + \alpha_2 + \mu_r) E_r \\
\frac{dI_r}{dt} &= \alpha_2 E_r - (\alpha_r + \mu_r + \delta_r) I_r \\
\end{align*}
\]

(4)

The description of each of the parameters in model (4) is illustrated in Table 1. Also, Fig. 1 depicts the transition between the several compartments taken into account in the model.

Our main interest is to examine a fractional form of the main model (4). Consider the fractional form of model (4) as

\[
\frac{^c D_t^q \psi(t)}{0} = \frac{1}{\Gamma(q)} \int_0^t (t - \tau)^{q-1} \psi(t) d\tau, \quad q \in (0, 1), t > 0.
\]

(1)

**Definition 2.** (see \[42,43\]). The Riemann-Liouville fractional derivative for a continuous function $\psi : (0, +\infty) \to \mathbb{R}$ is defined by

\[
\frac{^R D_t^q \psi(t)}{0} = \frac{1}{\Gamma(q)} \int_0^t (t - \tau)^{-q} \psi(t) d\tau, \quad q \in (0, 1), t > 0.
\]

(2)

**Definition 3.** (see \[42,43\]). The Caputo fractional derivative for a continuous function $\psi : (0, +\infty) \to \mathbb{R}$ is defined by

\[
\frac{^C D_t^q \psi(t)}{0} = \frac{1}{\Gamma(q)} \int_0^t (t - \tau)^{-q} \frac{d}{dt} \psi(t) d\tau, \quad q \in (0, 1), t > 0.
\]

(3)

Next, the formulation of the main problem under study will be derived in detail.

3. The formulation of the Monkeypox virus model based on the Caputo fractional derivative

This section is devoted to giving the main model that will be considered for studying the spread of the monkeypox virus, which can be in the following forms \[11\]. We suggest a Caputo fractional derivative model of the dynamics of monkeypox transmission that includes both human and rodent populations.
Next, we shall begin by investigating the equilibrium points and stability analysis of each point in the next section.

4. Equilibrium points, stability analysis, and basic reproduction number

4.1. Equilibrium points and stability analysis

The equilibrium points of the system can be obtained by equating the right-hand side of the system with zero. More specifically, the first equilibrium point is the DD equilibrium point, which corresponds to the disease-free equilibrium point for both human and rodent populations. It is expressed as follows $DD = \left( \frac{\mu_h}{\alpha}, 0, 0, 0, \frac{\delta_h}{\mu_h}, 0 \right)$. The second equilibrium point is the ED equilibrium point that corresponds to disease-free at rodents side and epidemic persistence for humans. It is obtained as follows:

$$ S_h = \frac{N_h(\alpha_h + \mu_h)(\gamma + \delta_h + \mu_h)}{\alpha_1 \beta_2}, $$

$$ E_h = \frac{N_h(\gamma + \delta_h + \mu_h)(\alpha + \phi + \delta_h + \mu_h)M}{\alpha_2 \beta_2(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}, $$

$$ M = (-N_h(\alpha_1 + \mu_h)(\gamma + \delta_h + \mu_h) + \alpha_1(\beta_1 \theta_3 - N_h(\gamma + \delta_h + \mu_h))) \mu_h, $$

$$ I_h = \frac{N_h(\alpha_1 \beta_2 \gamma)(\gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}{\alpha_1(\beta_1 \theta_3 \gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}, $$

$$ Q_h = \frac{N_h(\alpha_1 \beta_2 \gamma)(\gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}{\alpha_1(\beta_1 \theta_3 \gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}, $$

$$ R_h = \frac{N_h(\alpha_1 \beta_2 \gamma)(\gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}{\alpha_1(\beta_1 \theta_3 \gamma + \delta_h + \mu_h) - N_h(\alpha_1 \beta_2 \gamma + \delta_h + \mu_h)M}, $$

$$ T = (N_h(\alpha_1 + \mu_h)(\gamma + \delta_h + \mu_h) + \alpha_1(-\beta_1 \theta_3 + N_h(\gamma + \delta_h + \mu_h))) \mu_h, $$

$$ S_e = \frac{\theta_3}{\mu_h}, E_e = 0, I_e = 0. $$

Finally, the remaining two equilibrium points, denoted by DE and EE, represent the case where the values of the infected population take positive values for rodents while they take zero or positive values for the human population. They have a very complicated form and can be found by solving

$$ \theta_h - \left( \frac{\beta_1 \theta_3 + \beta_1 \theta_3}{N_h} \right) S_h - \mu_h S_h + \psi Q_h = 0, $$

$$ \left( \frac{\beta_1 \theta_3 + \beta_1 \theta_3}{N_h} \right) S_h - (\alpha_1 + \alpha_1 + \mu_h) E_h = 0, $$

$$ \alpha_1 E_h - (\mu_h + \gamma) I_h = 0, $$

$$ \alpha_2 E_h - (\phi + \gamma) I_h = 0, $$

$$ \gamma E_h - (\gamma + \delta_h + \mu_h) Q_h = 0, $$

$$ \theta_e - \left( \frac{\beta_1 \theta_3 + \beta_1 \theta_3}{N_h} \right) S_e - \mu_s S_e = 0, $$

$$ \left( \frac{\beta_1 \theta_3 + \beta_1 \theta_3}{N_h} \right) S_e - (\mu_e + \alpha_1) E_e = 0, $$

$$ \alpha_1 E_e - (\mu_e + \delta_e) I_e = 0. $$

symbolically or numerically.

In this section, we focus on DD and ED equilibrium points and also investigate associated stability regions in the space of parameters of the model.

The first step is to compute the Jacobian $J$ matrix at the DD equilibrium point and then express it as follows:

$$ J = \begin{pmatrix} -\mu_h & 0 & \frac{\beta_1 \theta_3}{N_h} & \phi & 0 & 0 & 0 & \frac{\beta_1 \theta_3}{N_h} \\ 0 & \alpha_1 - \beta_1 \theta_3 & -\mu_h & 0 & 0 & 0 & \frac{\beta_1 \theta_3}{N_h} \\ 0 & 0 & \gamma & \tau & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \end{pmatrix} $$

Also, the Jacobian matrix at the ED equilibrium point is evaluated as follows:

$$ J_1 = \begin{pmatrix} -\mu_h & 0 & \frac{\beta_1 \theta_3}{N_h} & \phi & 0 & 0 & 0 & \frac{\beta_1 \theta_3}{N_h} \\ 0 & \alpha_1 - \beta_1 \theta_3 & -\mu_h & 0 & 0 & 0 & \frac{\beta_1 \theta_3}{N_h} \\ 0 & 0 & \gamma & \tau & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_h & 0 & 0 & 0 \end{pmatrix} $$
The local asymptotic stability for a specific equilibrium point of the present fractional-order model with order \( q \) requires that all eigenvalues of the associated Jacobian matrix satisfy
\[
|\text{Arg}(\lambda)| > \frac{q\pi}{2}.
\]
Detailed numerical investigations are necessary to explore the influence of key parameters in the model on stability and to determine the regions of stability in the space of parameters. Without loss of generality \( N_h \) and \( N_r \) can be normalized to one. The following scenarios are not shown in the text but are illustrated in the figures.
considered to examine stability regions for the DD equilibrium point:

Firstly, the effects of the contact rate $\beta_2$ of humans and the natural death rate $\mu_h$ are examined in Fig. 2, where stability regions of the DD equilibrium point are colored in blue. The value of the recovery rate for humans is increased from $\gamma = 0.01$ in Fig. 2 (a) to $\gamma = 0.05$ and $\gamma = 0.1$ in Fig. 2 (b) and Fig. 2 (c), respectively. The other values of parameters are taken as

$$\theta_r = 0.2, \theta_h = 0.5, \alpha_1 = 0.2, \alpha_2 = 0.02, \alpha_3 = 0.027, \beta_1 = 0.000251, \beta_3 = 0.02, \mu = 0.02$$

$\delta_1 = 0.002, \delta_2 = 0.2, \phi = 2, r = 0.52, \gamma = 0.01, q = 0.9$.

The stability region for the disease-free equilibrium point increases with an increase in $\gamma$ values. In addition, for large values of contact rate between humans, the DD equilibrium point loses its stability.

Secondly, the influences of exposed to (not identified) infected human proportion $\alpha_1$ and exposed to highly infected and isolated human proportion $\alpha_2$ are examined in Fig. 3. In this case, the stability regions of the DD equilibrium point are colored in red. The value of the contact rate $\beta_2$ of humans are $\beta_2 = 0.2$ in Fig. 3 (a) and $\beta_2 = 0.1$ in Fig. 3 (b), respectively. It is clear that as $\beta_2$ decreases and the stability regions for DD are enlarged. Moreover, the value of the recovery rate for humans is increased from $\gamma = 0.001$ in Fig. 3 (b) to $\gamma = 0.01$ in Fig. 3 (c). It is found that the stability regions for DD are enlarged in this case too. The other values of parameters are taken as

$$\theta_r = 0.2, \theta_h = 0.5, \alpha_1 = 0.027, \beta_1 = 0.000251, \beta_3 = 0.027, \mu = 0.02$$

$\mu_h = 0.005, \delta_1 = 0.002, \delta_2 = 0.2, \phi = 2, r = 0.52, q = 0.8$.

Thirdly, the influences on the disease death rate $\delta_h$ along with the recovery rate are examined in Fig. 4. Here, the stability regions of the DD equilibrium point are colored green. The natural birth rate $\mu_h$ of humans is set as 0.00001, 0.01, and 0.1 in Fig. 4 (a, b, c), respectively, where the stability region of DD is depicted for each case. The other values of parameters are taken as

$$\theta_r = 0.2, \theta_h = 1.5, \alpha_1 = 0.002, \alpha_1 = 0.02, \alpha_3 = 0.027, \beta_1 = 0.000251, \beta_3 = 0.02$$

$\mu = 0.02, \delta_1 = 0.2, \phi = 2, r = 0.52, q = 0.7$.

Finally, the above scenarios are employed with the ED equilibrium point and the obtained results are shown in Figs. 5–7. It is demonstrated that the control measures to suppress the spread of the monkeypox epidemic should involve reducing the contact rate between humans in infected regions. In addition, sufficient support for the health care of infected individuals and funding of vaccination programs are crucial key factors in the battle against this pandemic.
4.2. Basic reproduction number

When analyzing an emerging epidemic, one of the critical points that arise is to employ a suitable mathematical model for estimating whether the infectious disease can spread within the population or not. The basic reproduction number is a critical parameter that can be used to answer the above question and determine the behavior of the epidemic when one infected case is introduced to a susceptible population. More specifically, the reproduction number $R_0$ is defined as the number of new infections resulting from a single infected individual during its infection time span. The well-known next-generation matrix technique is an efficient technique to compute $R_0$ in this work. For more details, see [44–46] and references therein.

The state variables $S_h, R_h, S_r, E_h, I_h, Q_h, R_h, S_r, E_r, I_r$ represent the disease-free state variables of the present model (5). On the other side, the state variables $E_h, I_h, Q_h, E_r, I_r$ represent the infected state variables of the model (5) and hence they constitute the infection class for the model. Now, the model system (5) can be expressed in the form

$$\frac{dX}{dt} = \Omega(X),$$

where

$$X = [S_h, E_h, I_h, Q_h, R_h, S_r, E_r, I_r]^T.$$
The first part of a vector field in the above system considers only the new infectious produced in the population, whereas the progression between infected or non-infected compartments is included in the second part of a vector field. Now, the transmission matrix $A$ and transition matrix $B$ are established as follows

$$A = \begin{pmatrix} 0 & \beta_2 & 0 & 0 & \beta_1 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_1 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$B = \begin{pmatrix} -\theta_1 + \left(\frac{\beta_1 I + \beta_2 S}{N_s}\right) S_i + \mu_i S_i - \phi Q_i \\ (\alpha_1 + \alpha_2 + \mu_i) E_i \\ -\alpha_1 E_i + (\mu_i + \delta_i + \gamma) I_i \\ -\alpha_2 E_i + (\phi + \tau + \delta_i + \mu_i) Q_i \\ -\gamma I_i - \tau Q_i + \mu_i R_i \\ -\theta_2 + \left(\frac{\beta_1 S_i L_i}{N_s}\right) S_i + \mu_i S_i + \phi Q_i \\ (\mu_i + \alpha_1) E_i - \alpha_1 E_i + (\mu_i + \delta_i) I_i \\ (\alpha_1 + \alpha_2 + \mu_i) S_i + \mu_i S_i \\ -\alpha_1 E_i + (\mu_i + \delta_i) I_i \end{pmatrix}$$

The stability regions for ED equilibrium point in $\mu_h - \beta_2$ plane of parameters when (a) $\gamma = 0.01$, (b) $\gamma = 0.05$ and (c) $\gamma = 0.1$.

$$\begin{array}{c}
\text{Fig. 5. Stability regions for ED equilibrium point in } \mu_h - \beta_2 \text{ plane of parameters when (a) } \gamma = 0.01, \text{ (b) } \gamma = 0.05 \text{ and (c) } \gamma = 0.1. \\
\end{array}$$

Consequently, the value of $R_0$ can be computed by

$$R_0 = \max \left\{ \frac{\alpha_1 \beta_2 (\alpha_1 + \alpha_2 + \mu_i) (\mu_i + \delta_i + \gamma)}{(\alpha_1 + \alpha_2 + \mu_i) (\mu_i + \delta_i + \gamma)}, \frac{\alpha_2 \beta_1 (\alpha_1 + \alpha_2 + \mu_i) (\mu_i + \delta_i + \gamma)}{(\alpha_1 + \alpha_2 + \mu_i) (\mu_i + \delta_i + \gamma)} \right\}.$$
public awareness, or even limited lockdowns can help in reducing the value of $R_0$ to be less than one. In addition, it is seen that increasing the value of $\alpha_2$, intensifying the tests for monkeypox virus within susceptible and exposed individuals, can reduce the value of $R_0$ and help control the spread of the monkeypox epidemic.

It is important to note that the points in the obtained stability regions for the DD equilibrium point coincide with the points which satisfy $R_0 < 1$ in the space of parameters.

5. Numerical simulation

This section describes the numerical method for obtaining the solution for the fractional-order Monkeypox virus described by the model (5). There are various numerical and analytical approaches for solving fractional-order systems, including the predictor-corrector method, the Homotopy method, and the Adomian decomposition method. Each of these methods has its advantages and drawbacks for obtaining the solutions to the fractional system (5). We choose to obtain the solutions using the predictor-corrector technique, which may adhere to some properties that may preserve the positivity of the obtained solution and be straightforward. This technique has been used multiple times to solve similar models [47]. The steps for solving model (5) can be summarized as follows:

**Step I**

We start with the first equation of (5). We obtain the following system for the fractional-order Monkeypox virus described by the model (5) as follows:

$$
\begin{align*}
S_1(t) &= S_1(0) + \int_0^t \frac{d}{dt} M_1(t, S_1, E_1, I_1, Q_1, R_1, S_2, E_2, I_2), \\
E_1(t) &= E_1(0) + \int_0^t \frac{d}{dt} M_2(t, S_1, E_1, I_1, Q_1, R_1, S_2, E_2, I_2), \\
I_1(t) &= I_1(0) + \int_0^t \frac{d}{dt} M_3(t, S_1, E_1, I_1, Q_1, R_1, S_2, E_2, I_2), \\
Q_1(t) &= Q_1(0) + \int_0^t \frac{d}{dt} M_4(t, S_1, E_1, I_1, Q_1, R_1, S_2, E_2, I_2), \\
R_1(t) &= R_1(0) + \int_0^t \frac{d}{dt} M_5(t, S_1, E_1, I_1, Q_1, R_1, S_2, E_2, I_2), \\
S_2(t) &= S_2(0) + \int_0^t \frac{d}{dt} M_6(t, S_2, E_2, I_2, Q_2, R_2, S_1, E_1, I_1), \\
E_2(t) &= E_2(0) + \int_0^t \frac{d}{dt} M_7(t, S_2, E_2, I_2, Q_2, R_2, S_1, E_1, I_1), \\
I_2(t) &= I_2(0) + \int_0^t \frac{d}{dt} M_8(t, S_2, E_2, I_2, Q_2, R_2, S_1, E_1, I_1).
\end{align*}
$$

(9)

Then, by applying the predictor-corrector method to the system (9), the integral terms are approximated and reduced to the following form. where $M_k, k \in \{1, 2, \ldots, 8\}$ are defined by Eq. (6) and the initial values

![Fig. 6. Stability regions for ED equilibrium point in $\alpha_1 - \alpha_2$ plane of parameters when (a) $\beta_2 = 0.2, \gamma = 0.001$, (b) $\beta_2 = 0.1, \gamma = 0.001$ and (c) $\beta_2 = 0.1, \gamma = 0.01$.](image)
\[\begin{align*}
S_0(t) &= S_0(0) + h^n \left[ p_1 M_1(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
E_0(t) &= E_0(0) + h^n \left[ p_1 M_2(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
I_0(t) &= I_0(0) + h^n \left[ p_1 M_3(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
Q_0(t) &= Q_0(0) + h^n \left[ p_1 M_4(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
R_0(t) &= R_0(0) + h^n \left[ p_1 M_5(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
S_1(t) &= S_1(0) + h^n \left[ p_1 M_6(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
E_1(t) &= E_1(0) + h^n \left[ p_1 M_7(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right], \\
I_1(t) &= I_1(0) + h^n \left[ p_1 M_8(0) + \sum_{i=1}^{n-1} p_i M_i(0) + \sum_{i=1}^{n-1} \Psi_{M_i} M_i \left( t, S_0, E_0, I_0, Q_0, R_0, S_0, E_0, I_0 \right) + \Psi_M M (t, S_0', E_0', I_0', Q_0', R_0', S_0', E_0', I_0') \right].
\end{align*}\]

Fig. 7. Stability regions for ED equilibrium point in \(\delta_0 - \gamma\) plane of parameters when (a) \(\mu_0 = 0.00001\), (b) \(\mu_0 = 0.01\) and (c) \(\mu_0 = 0.1\).
Fig. 8. Dynamical behavior of human state variables for case I.
Fig. 9. 3D plots of human populations versus time and fractional order $q$ with the corresponding contour plots for case I.
for $M_i$ can be evaluated in the form

$$M_i(0) = M_i(S_i(0), E_i(0), J_i(0), Q_i(0), R_i(0), S_i(0), E_i(0), J_i(0), k), k \in \{1, 2, \ldots, 8\},$$

and

$$n_i = \frac{(n-1)^q - n^q(n-q-1)}{\Gamma(2+q)},$$

$$\psi_{n}^{i} = \frac{(n-1)^{q+1} - 2n^{q+1} + n^{q} (n+1)}{\Gamma(2+q)}, n = 1, 2, 3, \ldots,$$

$$\psi_{1}^{i} = \frac{1}{\Gamma(2+q)}$$

Step II

Next, the predictors can be calculated as

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$S_i^{\rho}(t) = S_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$E_i^{\rho}(t) = E_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$I_i^{\rho}(t) = I_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$Q_i^{\rho}(t) = Q_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n+1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

$$R_i^{\rho}(t) = R_i(0) + h^\rho \sum_{i=1}^{n-1} \psi_{n-1}^{i} M_i(t, S_i, E_i, J_i, Q_i, R_i, S_i, E_i, I_i),$$

Fig. 10. Dynamical behavior of rodent state variables for case 1.
Following the predictors from Eq. (13), the solution for different state variables for both humans and rodents can be obtained. This technique possesses the property of providing a stable and convergent solution, as presented and proved by Garrappa et al. [47,48].

Next, we shall investigate the application of the proposed algorithm in two cases for each equilibrium point. The first case will give the numerical simulations and results for the disease-free equilibrium point (DD), while case II will give the results for the endemic equilibrium (EE) point.

Case I. DD Equilibrium point.

The initial values for the state variables for this case are chosen as \( S_h(0) = 0.7, E_h(0) = 0.1, I_h(0) = 0.1, Q_h(t) = 0.1, R_h(t) = 0.5, S_r(t) = 0.8, E_r(t) = 0.1, \) and \( I_r(t) = 0.1. \) The main system parameters are selected as \( \theta_r = 0.02, \theta_h = 0.015, \alpha_1 = 0.2, \alpha_2 = 0.02, \alpha_3 = 0.027, \beta_1 = 0.000251, \beta_2 = 0.01, \beta_3 = 0.1, \mu_r = 0.02, \mu_h = 0.015, \delta_h = 0.002, \delta_r = 0.2, \phi = 2, \tau = 0.52, \) and \( \gamma = 0.01. \) We examine the effect of different values of fractional order \( q \) on the human and rodent populations and their state variables. The effect of altering the order of the fractional derivative on the human population’s behaviors is exhibited in Fig. 8. In addition, a 3D plot of human populations versus time and fractional order \( q \) and the corresponding contour plot are exhibited in Fig. 9. It can be noticed from these figures that the choice of certain values for \( q = 0.9, 0.8, \) and \( 0.7 \) are in good agreement with the results for \( q = 1 \), proving that the proposed technique is effective in providing accurate results. The susceptible state \( S_h \) can be seen to have increasing behavior as time marches towards a stable state. The other states have decreasing behavior as time increased, proving that the disease may diminish over time, predicting the end of the pandemic. This verification can also be extended through the simulation of the rodent population in Fig. 10 for the three state variables along with their 3D and contour plots in Fig. 11. We conclude from these figures that the system is sensitive to the change in the fractional-order \( q \) and show how dependent it is on the history of the monkeypox virus system.

Case II. ED Equilibrium point.
The initial values for the state variables for this case are chosen as $S_h(0) = 0.16$, $E_h(0) = 0.03$, $I_h(0) = 0.6$, $Q_h(t) = 0.101$, $R_h(t) = 0.13$, $S_r(t) = 0.8$, $E_r(t) = 0.1$, and $I_r(t) = 0.1$. The main system parameters are selected as $\theta_r = 0.02$, $\theta_h = 0.005$, $\alpha_1 = 0.2$, $\alpha_2 = 0.06$, $\alpha_3 = 0.027$, $\beta_1 = 0.000251$, $\beta_2 = 0.1$, $\beta_3 = 0.027$, $\mu_r = 0.02$, $\mu_h = 0.005$, $\delta_h = 0.002$, $\delta_r = 0.2$, $\phi = 2$, $\tau = 0.52$, and $\gamma = 0.001$. The effect of changing the fractional order for the state variables of the ED case for different human state variables can be witnessed in Fig. 12. The observed behavior from this figure reflects the endemic state through the decreasing behavior as time is increased while the fractional-order $q$ is decreased. The 3-D plot of these state variables along with their contour plot is demonstrated in Fig. 13 for the human population. For the rodent population, the dynamics of the three state variables can be observed in Fig. 14. It can be noticed that the change in the fractional-order $q$ fits well with the results.
Fig. 13. 3D plots of human populations versus time and fractional order $q$ with the corresponding contour plots for case II.
for \( q = 1 \), which may indicate that the method is effective. The susceptible state \( S_r(t) \) is seen to have increasing behavior with time, reaching a state of stability, while the other two states are observed to be decreasing. The 3-D plot of the surfaces of all of the states is shown in Fig. 15 along with its contour plot.

Finally, it is worth noting that all simulations for this work were run on Windows 10, version 21H1, with a processor Intel(R) Core i5 CPU M520 @ 2.40 GHz and 4 GB of RAM. The CPU time of computation for all the results of the four cases \( (q = 1, q = 0.9, q = 0.8, q = 0.7) \) of Case I is approximately 1418 s, and the CPU time of computation for all the results of the four cases \( (q = 1, q = 0.9, q = 0.8, q = 0.7) \) of Case II is approximately 1435 s. Note that the step size = 0.005; start time = 0; and final time = 7000.

6. Conclusion

In this work, we investigate the transmission dynamics of a new Caputo fractional-order nonlinear model simulating the spread of the monkeypox virus. The proposed model examined the interaction between the human and rodent populations through eight mutually exclusive compartments. The human population is divided into five different states named the susceptible \( S_h(t) \), exposed \( E_h(t) \), infected \( I_h(t) \), isolated \( Q_h(t) \), and recovered \( R_h(t) \). The rodent population is classified into three categories, including susceptible \( S_r(t) \), exposed \( E_r(t) \), infected \( I_r(t) \). The reproduction number for the system is derived. In addition, the two types of equilibrium points, e.g., disease-free and endemic types, are investigated. The stability conditions and plotted stability regions for both the equilibrium points are presented. Numerical simulations are performed to verify the correctness of stability analysis of equilibrium points via using a predictor-corrector technique. The obtained results confirm the validity of stability analysis, reveal the effects of different parameters in the model, and help decide the most suitable way to prevent virus spread. It is observed that when memory influences are considered for the present model, through Caputo fractional-order derivatives, they affect the speed and time taken by solution trajectories towards steady-state equilibria. There are some key modifications to the present model which can be considered in future work. First, the influences of potential monkeypox vaccines can be included in the model. Second, the more realistic nonlinear incidence rates for the monkeypox virus can also be taken into account. Moreover, the spatio-temporal evolution of the epidemic can be examined in future work by employing reaction-diffusion PDEs in the model. Finally, the investigation of efficient control schemes, like optimal, sliding-mode, or adaptive controls to suppress the spread of monkeypox disease, can be conducted.

Availability of data and materials

All data generated or analyzed during this study are included in this article.

CRediT authorship contribution statement

A. El-Mesady: Conceptualization, Methodology, Formal analysis, Software, Data curation, Writing – original draft, Writing – review &
Declaration of competing 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 this paper.

Data availability

No data was used for the research described in the article.

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