# PAPER • OPEN ACCESS

# Fractional order mathematical model of monkeypox transmission dynamics

To cite this article: Olumuyiwa James Peter et al 2022 Phys. Scr. 97 084005

View the article online for updates and enhancements.

# You may also like

- <u>Analysis and Prediction of Subway</u> <u>Settlement Deformation Based on Grey</u> <u>Model</u> Zheng Shufen and Wen Jing
- <u>Monthly Runoff Prediction Using Wavelet</u> <u>Transform and Fast Resource</u> <u>Optimization Network (Fron) Algorithm</u> Jiahai Lu and Aiyun Li
- <u>Modeling of monolayer charge-stabilized</u> <u>colloidal crystals with static hexagonal</u> <u>crystal lattice</u>

A N Nagatkin and P E Dyshlovenko

# **Physica Scripta**

#### PAPER

CrossMark

#### **OPEN ACCESS**

RECEIVED 8 March 2022

REVISED 28 June 2022

ACCEPTED FOR PUBLICATION 6 July 2022

PUBLISHED 15 July 2022

licence

and DOI.

(cc

Original content from this work may be used under the terms of the Creative Commons Attribution 4.0

this work must maintain

٢

attribution to the author(s) and the title of the work, journal citation

Keywords: monkeypox disease, fractional-calculus, mathematical model, reproduction parameter, parameter estimation, stability analysis, Any further distribution of tuberculosis model

#### Abstract

In this paper, we present a deterministic mathematical model of monkeypox virus by using both classical and fractional-order differential equations. The model includes all of the possible interactions that contribute to disease spread in the population. We investigate the model's stability results in the disease-free case when  $R_0 < 1$ . When  $R_0 < 1$ , we show that the model is stable, otherwise it is unstable. To obtain the best fit that describes the dynamics of this disease in Nigeria, the model is fitted using the nonlinear least square method on cumulative reported cases of monkeypox virus from Nigeria between January to December 2019. Furthermore, adequate conditions for the existence and uniqueness of the solution of the model have been proved. We run numerous simulations of the proposed monkeypox model with varied input parameters to investigate the intricate dynamics of monkeypox infection under the effect of various system input parameters. We investigate the system's dynamical behavior to develop appropriate infection control policies. This allows the public to understand the significance of control parameters in the eradication of monkeypox in the population. Lowering the order of fractional derivatives has resulted in significant modifications. To the community's policymakers, we offered numerous parameters for the control of monkeypox.

# 1. Introduction

Monkeypox is a zoonotic disease that causes the monkeypox virus. Although it has been spotted elsewhere in the world, it is primarily found in Africa. Two outbreaks of a disease resembling the pox in groups of monkeys used for a study led to the discovery of monkeypox in 1958. Since January 2022, several countries have reported cases of monkeypox to the World Health Organization (WHO). As of June 15, 2022, 2103 cases with laboratory confirmation including one death had been reported to WHO [1, 2].

Direct contact with an infectious rash, scabs, or bodily fluids are other ways the virus can be transferred from one person to another, transmission is also possible through respiratory secretions during prolonged face-toface contact or during sexual activity or another intimate physical contact [1, 3, 4].

Primarily, wild animals like African rats and monkeys transmit the virus to people. However, human-tohuman transmissions are also frequent in most of the reported cases. Animal to humans occurs as a result of bites or scratches, the processing of bush meat, direct contact with bodily fluids, or consuming food contaminated by rodents. Direct contact with sores and bodily fluids from infected patients can spread the

# Fractional order mathematical model of monkeypox transmission dynamics

# Olumuyiwa James Peter<sup>1</sup> , Festus Abiodun Oguntolu<sup>2</sup>, Mayowa M Ojo<sup>3,4</sup>, Abdulmumin Olayinka Oyeniyi<sup>5</sup>, Rashid Jan<sup>6</sup> and Ilyas Khan<sup>7,\*</sup>

- Department of Epidemiology and Biostatistics, School of Public Health, University of Medical Sciences, Ondo City, Ondo State, Nigeria
- Department of Mathematics, Federal University of Technology, Minna Niger State, Nigeria
- Department of Mathematical Sciences, University of South Africa, Florida, South Africa
- Microbiology Division, Thermo Fisher Scientific, Lenexa, KS, United States of America
- African Institute for Mathematical Sciences, South West Region, Crystal Garden, P.O. Box 608 Limbe, Cameroon
- Department of Mathematics, University of Swabi, Swabi, KPK, Pakistan
- Department of Mathematics, College of Science Al-Zulfi Majmaah University, Al-Majmaah 11952, Saudi Arabia
- Author to whom any correspondence should be addressed.

#### E-mail: i.said@mu.edu.sa

infection. Researchers are investigating alternative ways the disease can spread, either through semen and vaginal fluids or in respiratory droplets [1, 5].

Monkeypox symptoms range from person to person. Fever, headache, muscle aches, backaches, swollen lymph nodes, chills, and exhaustion are among the typical symptoms of monkeypox. Most times, the symptoms of monkeypox are usually a moderate illness and most people recover on their own within a few weeks. Those with weak immune systems may have severe symptoms [1, 6]. The smallpox vaccine, antivirals, and vaccine immune globulin developed to protect against smallpox can be used as an alternative to prevent the spread of monkeypox but there is currently no proven, secure treatment for monkeypox virus infection. The vaccination is currently unavailable because smallpox has been eradicated worldwide [7, 8].

The disease has attracted little attention in the past, which has contributed to a lack of understanding of its transmission mechanisms. Despite this, quite a few researchers have attempted to use mathematical analysis to study the dynamics of the Monkeypox virus. [9] developed and analyzed a deterministic mathematical model for the Monkeypox virus. Their findings suggest that isolating infected individuals in the human population helps to reduce disease transmission. Also, [10] established a mathematical model for the dynamics of monkeypox transmission and presented it as a system of nonlinear differential equations. According to the numerical simulation, people's immune status influences how they recover after being infected with the orthopoxvirus. Numerous mathematical models on infectious disease have been studied in order to gain a better understanding of the transmission dynamics and different techniques to controlling the endemic disease (see [9, 11–17] for examples).

To better understand the dynamics of Monkeypox, [18] developed a mathematical model. The findings suggest that Monkeypox is under control and can be eradicated in a semi-endemic equilibrium through vaccination. Monkeypox, on the other hand, cannot be eradicated by vaccination alone in a fully endemic equilibrium. Furthermore, in the study of [19], the numerical simulations performed on the model revealed that the infectious individuals in the human and non-human primate populations will die out during the study period as a result of the proposed interventions. Researchers from several areas of science and engineering have recently expressed an interest in modeling with fractional differential equations, particularly mathematical modeling in epidemiology. The memory effect is one of the intriguing aspects of fractional-order models that cannot be found in classical differential equations due to the various properties of fractional differential equations, see for more details [20–23]. Several researchers have employed fractional differential equations to represent various infectious and non-infectious diseases in the recent age. The COVID-19 infection is one of these research topics that has been extensively examined and yielded useful results. [24-27] considers a fractional model for COVID 19 based on the Caputo derivative. A fractional-order model has been used to study tuberculosis with endogenous reactivation and exogenous reinfections [28]. A fractional-order HIV/AIDS epidemic model with Mittag-Leffler kernel was also studied in [29]. Other models with fractional order approach can be found in [30-33]. We first discuss the proposed model in detail in the integer order, and extend the model into fractional-order and obtain the necessary results.

After reviewing several works on the monkeypox virus and its mechanisms of transmission, to the best of our knowledge, this is the first work on the dynamics of the monkeypox virus with the fractional-order calculus. This work aims to study the transmission dynamics and control of monkeypox in the population using a classical and fractional-order model and to visualize how the memory index or fractional order parameter affects the dynamics of monkeypox disease and to know if it may be used as control parameter or not. The remainder of this script is arranged as follows: the model formulation and analyses of the monkeypox classical model are presented in section 2, while the data fitting and parameter estimation are performed in section 3. In section 4, the extension of the classical model to fractional-order and its results are presented. The analytical and numerical analysis of the fractional derivative solution is reported in section 5, while the conclusion is given in section 6.

# 2. Mathematical model and formulation

In this section, we develop a deterministic mathematical model of the monkeypox virus using some assumptions by considering the transmission of monkeypox which involves humans and the rodents population. We considered eight compartmental models; five human compartments and three rodent compartments.

The number of susceptible humans changes as a result of recruitment through birth rate or immigration  $\phi_h$ . It's decreased by natural death per capita rate  $\mu_h$  and force of infection  $\lambda_h$ . Hence, the rate of change in the number of susceptible humans is:

$$S'_h = \phi_h - (\lambda_h + \mu_h) S_h$$



The number of exposed humans is modeled by the force of infection  $\lambda_h$ . It's decreased by the rate  $\beta_h$  at which they become infectious and the natural death per capita rate  $\mu_h$ . Thus, the rate of change in the number of exposed humans is:

$$E_h' = \lambda_h S_h - (\mu_h + \beta_h) E_H$$

The number of Infectious humans increases with the rate of  $\beta_h E_H$  as a result of the transition from Exposed to Infectious. Thus, decreasing by natural death per capita rate  $\mu_h$ , disease-induced death  $\delta_1$ , the rate at which they recovered  $\omega$  and transit to clinically ill with the rate  $\gamma_h$ . So, the rate of change in the number of infectious humans is:

$$I'_{h} = \beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1})I_{h}$$

The number of clinically ill human increase as a result of those that need medical attention after being ill at the rate  $\gamma$ . These individuals transit from the infectious compartment to the recovery state with the rate  $\rho$ . it's decreased by natural per capita death  $\mu_h$ , disease-induced death  $\delta_2$ . Therefore, the rate at which the number of clinically ill human changes is given by:

$$C_h' = \gamma I_h - (\rho_h + \mu_h + \delta_2) C_h$$

The number of recovered human population increases as a result of the transition from being clinically ill to recovered compartments at the rate  $\rho$ . Then decreased by natural per capita death  $\mu_h$ , those that recovered from being infectious due to natural immunity with the rate  $\omega_H$ . Thus, the rate of change in the population of recovered humans is:

$$R_h' = \rho C_h + \omega I_h - \mu_h R_h$$

The number of susceptible rodents changes as a result of recruitment through constant birth rate  $\phi_r$ . They become infected by interaction with infected rodents which is modeled by  $\frac{\sigma I_r}{N_r}$ . It's decreased by natural death per capita rate  $\mu_r$ . Hence, the rate of change in the population of susceptible rodents is:

$$S_r' = \phi_r - \frac{\sigma I_r}{N_r} S_r - \mu_r S_r$$

The number of Exposed rodent increases with force of infection  $\lambda_r = \frac{\sigma I_r}{N_r}$ . It's decreased by the rate  $\epsilon$  at which they transit to infectious class and natural death per capita rate  $\mu_r$ . Hence, the rate of change in the population of exposed rodents is:

$$E_r' = \lambda_r S_r - (\epsilon E_r + \mu_r) I_r$$

The number of Infectious rodents increases with the rate  $\epsilon$  at which exposed rodents become infectious. It's decreased by natural death per capita rate  $\mu_r$ . Hence, the rate of change in the population of infected rodents is:

$$I_r' = \epsilon E_r - \mu_r I_R$$

Therefore, based on the above descriptions, the corresponding mathematical equations are given by a system of non-linear ordinary differential equations below:

Table 1. V	Variables and	description.
------------	---------------	--------------

Variables	Description	
$\overline{S_h(t)}$	Number of susceptible human	
$E_h(t)$	Number of exposed human	
$I_h(t)$	Number of infectious human	
$C_h(t)$	Number of clinically ill human	
$R_h(t)$	Number of recovered human	
$S_r(t)$	Number of susceptible rodent	
$E_r(t)$	Number of exposed rodent	
$I_r(t)$	Number of infected rodent	
Parameters	Description	
$\phi_h$	Recruitment into susceptible human class.	
$\phi_r$	Recruitment into susceptible rodent class.	
$\mu_h$	Per capita natural death rate in humans	
$\mu_r$	Per capita natural death rate in rodent	
β	Disease progression rate from exposed to infectious	
	humans	
$\delta_1$	Infectious human disease-induced death rate	
$\delta_2$	Clinically ill human disease-induced death rate	
$\gamma$	Clinically ill rate	
ρ	Recovery rate of Clinically ill human	
ω	Natural recovery rate due to immunity	
θ	Progression rate from exposed rate to infected rate	
$\alpha_1$	Contact rate between infected human and suscep- tible human	
$\alpha_2$	Contact rate between infected rats and susceptible humans	
σ	Contact rate between infected rats and susceptible rodents	
$\lambda_{L}$	Force of infection	

$$\begin{cases} S'_{h} = \phi_{h} - (\lambda_{h} + \mu_{h})S_{h} \\ E'_{h} = \lambda_{h}S_{h} - (\mu_{h} + \beta)E_{h} \\ I'_{h} = \beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1})I_{h} \\ C'_{h} = \gamma I_{h} - (\rho + \mu_{h} + \delta_{2})C_{h} \\ R'_{h} = \rho C_{h} + \omega I_{h} - \mu_{h}R_{h} \\ S'_{r} = \phi_{r} - \lambda_{r}S_{r} - \mu_{r}S_{r} \\ E'_{r} = \lambda_{r}S_{r} - (\epsilon + \mu_{r})E_{r} \\ I'_{r} = \epsilon E_{r} - \mu_{r}I_{r} \end{cases}$$
(1)

Given the force of infection  $\lambda_h = \frac{\alpha_1 I_h}{N_h} + \frac{\alpha_2 I_r}{N_r}$  such that:  $\frac{I_h}{N_h}$  is the proportion of infected human and  $\alpha_1$ , the rate at which susceptible human interact with infected human.  $\frac{I_r}{N_r}$  is the proportion of infected rodent and  $\alpha_2$ , the rate at which susceptible human interact with infected rodent.

The compartmental model in figure 1 represent the transmission of Monkey Pox.

The state variables and parameters are described in table 1.

#### 2.1. Positivity and boundedness of the solution

**Theorem 1.** Let  $S_h(0) = S_{h_0}$ ,  $E_h(0) = E_{h_0}$ ,  $I_h(0) = I_{h_0}$ ,  $C_h(0) = C_{h_0}$ ,  $R_h(0) = R_{h_0}$ ,  $S_r(0) = S_{r_0}$ ,  $E_r(0) = E_{r_0}$ and  $I_r(0) = I_{r_0}$  be the initial values of the state variables. If  $S_{h_0}$ ,  $E_{h_0}$ ,  $I_{h_0}$ ,  $C_{h_0}$ ,  $R_{r_0}$  and  $S_{r_0}$  are positive then it implies that  $S_h(t)$ ,  $E_h(t)$ ,  $I_h(t)$ ,  $C_h(t)$ ,  $R_h(t)$ ,  $S_r(t)$ ,  $E_r(t)$  and  $I_r(t)$  are positive for all time t > 0. However,

$$t \xrightarrow{\lim} \infty \sup N_h(t) \leq \frac{\phi_h}{\mu_h} and t \xrightarrow{\lim} \infty \sup N_r(t) \leq \frac{\phi_r}{\mu_r}$$

Also, if  $N_{h_0} \leq \frac{\phi_h}{\mu_h}$ , then  $N_h(t) \leq \frac{\phi_h}{\mu_h}$ , if  $N_{r_0} \leq \frac{\phi_r}{\mu_r}$  then  $N_r(t) \leq \frac{\phi_r}{\mu_r}$  then, the feasible domain for the differential equation are given by

$$\eta_{N_h} = \left\{ (S_h, E_h, I_h, C_h, R_h) \subset \mathbb{R}^5_+ : S_h + E_h + I_h + C_h + R_h \leqslant \frac{\phi_h}{\mu_h} \right\}$$
$$\eta_{N_r} = \left\{ (S_r, E_r, I_r) \subset \mathbb{R}^3_+ : S_r + E_r + I_r \leqslant \frac{\phi_r}{\mu_r} \right\}$$

such that

$$\eta = \eta_{N_h} \times \eta_{N_r} \subset \mathbb{R}^5_+ \times \mathbb{R}^3_+$$

and we have that  $\eta$  is positive invariant.

**Proof.** Let  $S_{h_0}$ ,  $E_{h_0}$ ,  $I_{h_0}$ ,  $C_{h_0}$ ,  $R_{h_0}$ ,  $S_{r_0}$ ,  $E_{r_0}$  and  $I_{r_0}$  be positive, we want to show that the state variables are also positive. From system (1) we have:

 $S_h' = \phi_1 - (\lambda_h + \mu_h)S_h$ 

then,

$$S_h' + (\lambda_h + \mu_h)S_h = \phi_h$$

Since  $\phi_h \ge 0$  it follows that,

 $S'_h + (\lambda_h + \mu_h) S_h \ge 0.$ 

Now, we have:

 $\frac{dS_h}{S_h} \ge -(\lambda_h + \mu_h)dt \tag{2}$ 

By integrating equation(2) we have:

$$\ln(S_h) \ge -\int (\lambda_h + \mu_h) dt + c.$$

Let  $A(t) = -\int (\lambda_h + \mu_h) dt$ , it implies that

$$\ln(S_h(t)) \ge A(t) + c \tag{3}$$

at t = 0 we have

$$\ln(S_h(0)) \ge A(0) + c \tag{4}$$

We subtract equations (4) from (3) to have

$$\ln\left(\frac{S_h(t)}{S_h(0)}\right) \geqslant A(t) - A(0)$$

Taking the exponential of both sides, we have:

$$S_h(t) \ge S_h(0)e^{A(t)-A(0)} \ge S_h(0) \text{ for } t \ge 0$$

Since  $S_h(0) = S_{h_0}$  is positive, that is,  $S_{h_0} > 0$  for t > 0 it implies that

$$S_h(t) \ge S_h(0) > 0$$
 fort > 0.

We have  $S_h(t) > 0$  for all t > 0 and we conclude that  $S_h(t)$  is non-negative for all t > 0. Hence, we have  $S_h > 0$ . This is true in the same way for the other state variables. This shows that  $S_h(t)$ ,  $E_h(t)$ ,  $I_h(t)$ ,  $C_h(t)$ ,  $R_h(t)$ ,  $S_r(t)$ ,  $E_r(t)$  and  $I_r(t)$  are positive for all time t > 0.

To describe the boundedness of solution, we use the theorem below:

**Theorem 2.** Given a positive set of solutions  $(S_h(t), E_h(t), I_h(t), C_h(t), R_h(t), S_r(t), E_r(t), I_r(t))$ , there exists a domain  $\eta$  in which this solution set is contained and bounded.

**Proof.** The total population for human is given by

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + C_h(t) + R_h(t)$$

Such that

$$N'_{h}(t) = S'_{h}(t) + E'_{h}(t) + I'_{h}(t) + C'_{h}(t) + R'_{h}(t)$$

By simplification we have

$$\frac{dN_h}{dt} = \phi_h - \mu_h (S_h + E_h + I_h + C_h + R_h) - (\delta_1 I_h + \delta_2 C_h).$$

But  $N_h = S_h + E_h + I_h + C_h + R_h$ . So, we have

$$\frac{dN_h}{dt} = \phi_h - \mu_h N_h - (\delta_1 I_h + \delta_2 C_h) = \frac{dN_h}{dt} - (\phi_r - \mu_h N_h) = -(\delta_1 I_h + \delta_2 C_h)$$

since  $I_h$  and  $C_h$  are positive, we have  $-(\delta_1 I_h + \delta_2 C_h) \leq (\delta_1 I_h + \delta_2 C_h)$ . It implies that:

$$\frac{dN_h}{dt} - (\phi_h - \mu_h N_h) = -(\delta_1 I_h + \delta_2 C_h) \leqslant (\delta_1 I_h + \delta_2 C_h)$$

$$\frac{dN_h}{dt} - (\phi_h - \mu_h N_h) \leqslant (\delta_1 I_h + \delta_2 C_h)$$
(5)

In the absence of Monkey Pox in the population ( $I_h = C_h = 0$ ), inequality (5) implies that:

$$egin{aligned} rac{dN_h}{dt} - (\phi_h - \mu_h N_h) \leqslant 0, &= rac{dN_h}{dt} \leqslant (\phi_h - \mu_H N_h), \ &= rac{dN_h}{(\phi_h - \mu_h N_h)} \leqslant dt \end{aligned}$$

Integrate both sides form t = 0 and  $t = t_*$ , we have

$$\int_0^{t_*} \frac{dN_h}{(\phi_h - \mu_h N_h)} \leqslant \int_0^{t_*} dt$$

we have

$$\left[-\frac{1}{\mu_h}\ln(\phi_h-\mu_hN_h)\right]_0^{t_*} \leqslant [t]_0^{t_*}$$

This gives us

$$\ln(\phi_h - \mu_h N_h(t_*)) - \ln(\phi_1 - \mu_h N_h(0)) \ge -\mu_h t_*$$

then

$$\ln\left(\frac{\phi_h - \mu_h N_h(t_*)}{\phi_h - \mu_h N_h(0)}\right) \ge -\mu_h t_*$$

Taking exponential of both sides we have:

$$\begin{aligned} \frac{\phi_h - \mu_h N_h(t_*)}{\phi_h - \mu_h N_h(0)} &\ge e^{-\mu_h t_*} \\ \phi_h - \mu_h N_h(t_*) &\ge (\phi_h - \mu_h N_h(0)) e^{-\mu_h t_*} \end{aligned}$$

which gives

$$\mu_{h}N_{h}(t_{*}) \leqslant \phi_{h} - (\phi_{h} - \mu_{h}N_{h}(0))e^{-\mu_{h}t_{*}}$$

$$N_{h}(t_{*}) \leqslant \frac{\phi_{h}}{\mu_{h}} - \frac{(\phi_{h} - \mu_{h}N_{h}(0))}{\mu_{h}}e^{-\mu_{h}t_{*}}.$$
(6)

By taking the limit of inequality (6) as  $t_* \longrightarrow \infty$ , we have

$$N_h(t_*) \leqslant rac{\phi_h}{\mu_h}$$

Thus, we have  $N_h(t) \leq \frac{\phi_h}{\mu_h}$ .

Which implies that  $N_h \in [0, \frac{\phi_h}{\mu_h}]$  that is  $\lim_{t \to \infty} \sup N_h(t) = \frac{\phi_h}{\mu_h}$  and if  $N_{h_0} \leq \frac{\phi_h}{\mu_h}$ , then  $N_h(t) \leq \frac{\phi_h}{\mu_h}$ . Thus,  $N_h$  is bounded. In the same way, we can demonstrate that the rodent population  $N_r(t)$  is bounded.

Therefore, this establishes the notion of  $\eta$  as required, that is, the solution to the model's equations are contained is positively invariant. So, we conclude that the model is epidemiologically feasible and well-posed in  $\eta$ .

#### 2.2. Monkey Pox-Free Equilibrium (MFE)

The Monkey Pox-Free equilibrium  $\Omega_{MFE} = (S_h^*, E_h^*, I_h^*, C_h^*, R_h^*, S_r^*, E_r^*, I_r^*)$  is defined as the point at which no disease is present in the population. All infected classes will be equal to zero.

Thus, the Monkey Pox-Free equilibrium satisfies

$$\Omega_{MFE} = (S_h^*, E_h^*, I_h^*, C_h^*, R_h^*, S_r^*, E_r^*, I_r^*) = \left(\frac{\phi_h}{\mu_h}, 0, 0, 0, 0, \frac{\phi_r}{\mu_r}, 0, 0\right).$$
(7)

#### 2.3. Monkey Pox Endemic Equilibrium (MEE)

Let  $\zeta_{MEE} = (S_h^{**}, E_h^{**}, I_h^{**}, C_h^{**}, R_h^{**}, S_r^{**}, E_r^{**}, I_r^{**})$  be defined as the point where there is Monkey Pox in the population. Here, all infected classes are not equal to zero. Consider the equations in system (1)

$$S_h^{**} = \frac{\phi_h}{\lambda_h + \mu_h} \tag{8}$$

$$E_h^{**} = \frac{\lambda_h}{\beta + \mu_h} S_h^{**} \tag{9}$$

By putting the value of  $S_h^{**}$  in (9) we have,

$$E_h^{**} = \frac{\lambda_h \phi_h}{(\beta + \mu_h)(\lambda_h + \mu_h)}.$$
(10)

Also,

$$I_h^{**} = \frac{\beta E_h^{**}}{\omega + \gamma + \delta_1 + \mu_h} \tag{11}$$

By putting the value of  $E_h^{**}$  in (9) we have,

$$I_h^{**} = \frac{\beta \lambda_h \phi_h}{(\omega + \gamma + \delta_1 + \mu_h)(\beta + \mu_h)(\lambda_h + \mu_h)}$$
(12)

Furthermore,

$$C_h^{**} = \frac{\gamma I_h^{**}}{\rho + \delta_2 + \mu_h} \tag{13}$$

By substituting the value of  $I_h^{**}$  in (13) we have,

$$C_h^{**} = \frac{\gamma \beta \lambda_h \phi_h}{(\rho + \delta_2 + \mu_h)(\omega + \gamma + \delta_1 + \mu_h)(\beta + \mu_h)(\lambda_h + \mu_h)}$$
(14)

Also,

$$R_{h}^{**} = \frac{\rho C_{h}^{**} + \omega I_{h}^{**}}{\mu_{h}}$$
(15)

By substituting the value of  $C_h^{**}$  and  $I_h^{**}$  in (15) we have,

$$R_{h}^{**} = \frac{\frac{\rho\gamma\beta\lambda_{h}\phi_{h}}{(\rho+\delta_{2}+\mu_{h})(\omega+\gamma+\delta_{1}+\mu_{h})(\beta+\mu_{h})(\lambda_{h}+\mu_{h})} + \frac{\omega\beta\lambda_{h}\phi_{h}}{(\omega+\gamma+\delta_{1}+\mu_{h})(\beta+\mu_{h})(\lambda_{h}+\mu_{h})}}{\mu_{h}}$$
(16)

Which give:

$$R_{h}^{**} = \frac{\rho\gamma\beta\lambda_{h}\phi_{h}\mu_{h}}{(\rho + \delta_{2} + \mu_{h})(\omega + \gamma + \delta_{1} + \mu_{h})(\beta + \mu_{h})(\lambda_{h} + \mu_{h})} + \frac{\omega\beta\lambda_{h}\phi_{h}\mu_{h}}{(\omega + \gamma + \delta_{1} + \mu_{h})(\beta + \mu_{h})(\lambda_{h} + \mu_{h})}$$
(17)

Similarly,

$$S_r^{**} = \frac{\phi_r}{\lambda_r + \mu_r}, \quad E_r^{**} = \frac{\lambda_r \phi_r}{(\epsilon + \mu_r)(\lambda_r + \mu_r)}, \quad I_r^{**} = \frac{\epsilon \lambda_r \phi_r}{(\epsilon + \mu_r)(\lambda_r + \mu_r)\mu_r}.$$

Hence, the Monkey Pox Endemic equilibrium satisfies

$$\begin{aligned} \zeta_{MEE} &= (S_h^{**}, E_h^{**}, I_h^{**}, C_h^{**}, R_h^{**}, S_r^{**}, E_r^{**}, I_r^{**}) \\ &= \left(\frac{\phi_h}{\lambda_h + \mu_h}, \frac{\lambda_h \phi_h}{(\beta + \mu_h)(\lambda_h + \mu_h)}, \frac{\beta \lambda_h \phi_h}{(\omega + \gamma + \delta_1 + \mu_h)(\beta + \mu_h)(\lambda_h + \mu_h)}, \frac{\gamma \beta \lambda_h \phi_h}{(\rho + \delta_2 + \mu_h)(\omega + \gamma + \delta_1 + \mu_h)(\beta + \mu_h)(\lambda_h + \mu_h)}, \end{aligned}$$

O J Peter et al

$$\frac{\rho\gamma\beta\lambda_h\phi_h\mu_h}{(\rho+\delta_2+\mu_h)(\omega+\gamma+\delta_1+\mu_h)(\beta+\mu_h)(\lambda_h+\mu_h)} + \frac{\omega\beta\lambda_h\phi_h\mu_h}{(\omega+\gamma+\delta_1+\mu_h)(\beta+\mu_h)(\lambda_h+\mu_h)}, \frac{\phi_r}{\lambda_r+\mu_r}, \frac{\lambda_r\phi_r}{(\epsilon+\mu_r)(\lambda_r+\mu_r)}, \frac{\epsilon\lambda_r\phi_r}{(\epsilon+\mu_r)(\lambda_r+\mu_r)\mu_r}.$$

### 2.4. Basic reproduction number

We will compute the reproduction number using the next generation matrix method. According to the principle of next generation matrix, the basic reproduction number is the spectral radius of the next generation matrix  $FV^{-1}$ [34]. That is the basic reproduction number

$$R_0 = \rho(FV^{-1}) \tag{18}$$

Therefore, using the equation (18) above, we split the differential equations into a new infection matrix F and transfer matrix between compartment V.

and

$$V = \begin{pmatrix} \beta + \mu_h & 0 & 0 & 0 & 0 \\ -\beta & \delta_1 + \gamma + \mu_h + \omega & 0 & 0 & 0 \\ 0 & -\gamma & \delta_2 + \mu_h + \rho & 0 & 0 \\ 0 & 0 & 0 & \epsilon + \mu_r & 0 \\ 0 & 0 & 0 & -\epsilon & \mu_r \end{pmatrix}$$

Such that:

$$V^{-1} = \begin{pmatrix} \frac{1}{\beta + \mu_h} & 0 & 0 & 0 & 0 \\ \frac{\beta}{(\beta + \mu_h)(\delta_1 + \gamma + \mu_h + \omega)} & \frac{1}{\delta_1 + \gamma + \mu_h + \omega} & 0 & 0 & 0 \\ \frac{\beta\gamma}{(\beta + \mu_h)(\delta_1 + \gamma + \mu_h + \omega)(\delta_2 + \mu_h + \rho)} & \frac{\gamma}{(\delta_1 + \gamma + \mu_h + \omega)(\delta_2 + \mu_h + \rho)} & \frac{1}{\delta_2 + \mu_h + \rho} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\epsilon + \mu_r} & 0 \\ 0 & 0 & 0 & \frac{\epsilon}{(\epsilon + \mu_r)\mu_r} & \frac{1}{\mu_r} \end{pmatrix}$$

Therefore,

In order to obtain the spectral radius, we need to calculate the eigenvalues of  $FV^{-1}$ . Therefore, by calculations and simplification we obtained the following eigenvalues:

$$\lambda = \left[\frac{\alpha_1\beta\phi_h}{N_h\mu_h^3 + (N_h\beta + N_h\delta_1 + N_h\gamma)\mu_h^2 + (N_h\beta\delta_1 + N_h\beta\gamma)\mu_h + (N_h\beta\mu_h + N_h\mu_h^2)\omega}, \frac{\epsilon\phi_r\sigma}{N_r\epsilon\mu_r^2 + N_r\mu_r^3}, 0, 0, 0\right]$$
$$= \left[\frac{\alpha_1\beta\phi_h}{N_h(\beta + \mu_h)(\delta_1 + \gamma + \mu_h + \omega)\mu_h}, \frac{\epsilon\phi_r\sigma}{N_r(\epsilon + \mu_r)\mu_r^2}, 0, 0, 0\right]$$

Therefore,

$$R_0 = \max\left\{\frac{\alpha_1\beta\phi_h}{N_h(\beta+\mu_h)(\delta_1+\gamma+\mu_h+\omega)\mu_h}, \frac{\epsilon\phi_r\sigma}{N_r(\epsilon+\mu_r)\mu_r^2}\right\}$$
(19)

Hence,

$$R_0 = \max\{R_0^h, R_0^r\}$$
(20)

We consider the following cases:

1. If 
$$\alpha_1 \beta \phi_h > N_h (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h$$
 and  $\epsilon \phi_r \sigma > N_r (\epsilon + \mu_r) \mu_r^2$ , then  $R_0 > 1$ 

- 2. If  $\alpha_1 \beta \phi_h < N_h(\beta + \mu_h)(\delta_1 + \gamma + \mu_h + \omega)\mu_h$  and  $\epsilon \phi_r \sigma > N_r(\epsilon + \mu_r)\mu_r^2$ , then  $R_0 > 1$
- 3. If  $\alpha_1 \beta \phi_h > N_h (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h$  and  $\epsilon \phi_r \sigma < N_r (\epsilon + \mu_r) \mu_r^2$ , then  $R_0 > 1$
- 4. If  $\alpha_1 \beta \phi_h < N_h (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h$  and  $\epsilon \phi_r \sigma < N_r (\epsilon + \mu_r) \mu_r^2$ , then  $R_0 < 1$ .

The Monkey Pox-free equilibrium (MFE) is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

#### 2.5. Stability of Monkey Pox-Free Equilibrium (MFE)

To determine the stability of the disease-free equilibrium, we compute the Jacobian matrix of the system at disease-free equilibrium, we carried out a linear stability analysis, that is, we compute the eigenvalues and the sign of the eigenvalues is used to determine the stability.

We have,

$$J = \begin{pmatrix} -(\lambda_h + \mu_h) & 0 & \frac{-\alpha_1 S_h}{N_h} & 0 & 0 & 0 & 0 & \frac{-\alpha_2 S_h}{N_r} \\ \lambda_h & -(\mu_h + \beta) & \frac{\alpha_1 S_h}{N_h} & 0 & 0 & 0 & 0 & \frac{\alpha_2 S_h}{N_r} \\ 0 & \beta & J_{33} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega & \rho & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\lambda_r + \mu_r) & 0 & \frac{-\sigma S_r}{N_r} \\ 0 & 0 & 0 & 0 & 0 & \lambda_r & -(\epsilon + \mu_r) & \frac{\sigma S_r}{N_r} \\ 0 & 0 & 0 & 0 & 0 & 0 & \epsilon & -\mu_r \end{pmatrix}$$

where

$$J_{33} = -(\omega + \gamma + \mu_h + \delta_1)$$
$$J_{44} = -(\rho + \mu_h + \delta_2)$$

Then, evaluating our Jacobian matrix at Monkey Pox-Free Equilibrium (MFE) we have:

$$J_{MFE} = \begin{pmatrix} -\mu_h & 0 & \frac{-\alpha_1 \phi_h}{N_h \mu_h} & 0 & 0 & 0 & 0 & \frac{-\alpha_2 \phi_h}{N_r \mu_h} \\ 0 & -(\mu_h + \beta) & \frac{\alpha_1 \phi_h}{N_h \mu_h} & 0 & 0 & 0 & 0 & \frac{\alpha_2 \phi_h}{N_r \mu_h} \\ 0 & \beta & J_{33} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega & \rho & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_r & 0 & \frac{-\sigma \phi_r}{N_r \mu_r} \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\epsilon + \mu_r) & \frac{\sigma \phi_r}{N_r \mu_r} \\ 0 & 0 & 0 & 0 & 0 & 0 & \epsilon & -\mu_r \end{pmatrix}$$

Now, we compute the eigenvalues and the characteristics polynomial which is given by  $|J_{MFE} - \lambda I|$  where *I* is a  $8 \times 8$  unit matrix, and the following values of  $\lambda$  were obtained:

$$\begin{split} \lambda_{1} &= -\frac{N_{r}\epsilon\mu_{r} + 2 N_{r}\mu_{r}^{2} + \sqrt{N_{r}^{2}\epsilon^{2}\mu_{r}^{2} + 4 N_{r}\epsilon\mu_{r}\phi_{r}\sigma}}{2 N_{r}\mu_{r}}, \\ \lambda_{2} &= -\frac{N_{r}\epsilon\mu_{r} + 2 N_{r}\mu_{r}^{2} - \sqrt{N_{r}^{2}\epsilon^{2}\mu_{r}^{2} + 4 N_{r}\epsilon\mu_{r}\phi_{r}\sigma}}{2 N_{r}\mu_{r}}, \\ \lambda_{3} &= -\frac{2 N_{r}\mu_{h}^{2} + N_{r}\mu_{h}\omega + (N_{r}\beta + N_{r}\delta_{1} + N_{r}\gamma)\mu_{h} + \sqrt{L}}{2 N_{r}\mu_{h}}, \\ \lambda_{4} &= -\frac{2 N_{r}\mu_{h}^{2} + N_{r}\mu_{h}\omega + (N_{r}\beta + N_{r}\delta_{1} + N_{r}\gamma)\mu_{h} - \sqrt{L}}{2 N_{r}\mu_{h}}, \\ \lambda_{5} &= -\delta_{2} - \mu_{h} - \rho, \\ \lambda_{6} &= -\mu_{r}, \\ \lambda_{8} &= -\mu_{h}, \end{split}$$

Where

$$\begin{split} L &= N_r^2 \mu_h^2 \omega^2 + 4 \, N_r \alpha_1 \beta \mu_h \phi_h \\ &- 2 \, (N_r^2 \beta - N_r^2 \delta_1 - N_r^2 \gamma) \mu_h^2 \omega + (N_r^2 \beta^2 - 2 \, N_r^2 \beta \delta_1 + N_r^2 \delta_1^2 + N_r^2 \gamma^2 - 2 \, (N_r^2 \beta - N_r^2 \delta_1) \gamma) \mu_h^2 \end{split}$$

These shows that six of the eigenvalues have a negative real parts. Hence, the Monkey Pox-free equilibrium is asymptotically stable if:

$$-\frac{N_r\epsilon\mu_r + 2N_r\mu_r^2 - \sqrt{N_r^2\epsilon^2\mu_r^2 + 4N_r\epsilon\mu_r\phi_r\sigma}}{2N_r\mu_r} < 0$$
$$-N_r\epsilon\mu_r - 2N_r\mu_r^2 + \sqrt{N_r^2\epsilon^2\mu_r^2 + 4N_r\epsilon\mu_r\phi_r\sigma} < 0$$
(21)

by simplifying (21), we have:

$$\frac{\epsilon\phi_r\sigma}{N_r(\epsilon+\mu_r)\mu_r^2} < 1 \tag{22}$$

and if:

$$-\frac{2 N_r \mu_h^2 + N_r \mu_h \omega + (N_r \beta + N_r \delta_1 + N_r \gamma) \mu_h - \sqrt{L}}{2 N_r \mu_h} < 0$$

$$\sqrt{L} < 2 N_r \mu_h^2 + N_r \mu_h \omega + (N_r \beta + N_r \delta_1 + N_r \gamma) \mu_h$$

$$4 N_r \alpha_1 \beta \mu_h \phi_h < -N_r^2 \mu_h^2 \omega^2 + 2 (N_r^2 \beta - N_r^2 \delta_1 - N_r^2 \gamma) \mu_h^2 \omega$$

$$- (N_r^2 \beta^2 - 2 N_r^2 \beta \delta_1 + N_r^2 \delta_1^2 + N_r^2 \gamma^2 - 2 (N_r^2 \beta - N_r^2 \delta_1) \gamma) \mu_h^2$$

$$+ (2 N_r \mu_h^2 + N_r \mu_h \omega + (N_r \beta + N_r \delta_1 + N_r \gamma) \mu_h)^2$$

$$4 N_r \alpha_1 \beta \mu_h \phi_h < 4 N_r^2 (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h^2$$

By simplification we have:

$$\frac{\alpha_1 \beta \phi_h}{N_r (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h} < 1.$$
<sup>(23)</sup>

Therefore, the Monkey Pox-free equilibrium (DFE) is asymptotically stable if

$$\begin{aligned} \frac{\epsilon \phi_r \sigma}{N_r (\epsilon + \mu_r) \mu_r^2} < 1 \quad and \\ \frac{\alpha_1 \beta \phi_h}{N_r (\beta + \mu_h) (\delta_1 + \gamma + \mu_h + \omega) \mu_h} < 1. \end{aligned}$$

## 3. Parameter estimation and model fitting

We use three distinct methods to collect our data. The formulated model (1) includes fourteen parameters, two of which were obtained from the literature. Two parameters are estimated based on the data from Nigeria, natural mortality rate and recruitment rate. The average lifespan in Nigeria is approximately 61 years [35]. The per capita natural death rate in human is estimated to be  $\mu_h = \frac{1}{61 \times 54}$  per weeks. We assume that the total

Table 2. Model	parameter va	lues
----------------	--------------	------

Parameter	Description	Value	Source
$\phi_h$	Recruitment into susceptible humans	64 850	Estimated
$\phi_r$	Recruitment into susceptible rodents	0.200 000	[36]
$\iota_h$	Mortality rate in humans	0.000303	Estimated
l <sub>r</sub>	Mortality rate in rodents	0.002000	[10]
3	Disease progression rate from expose to infectious humans	0.016744	Fitted
5 <sub>1</sub>	Infectious humans disease-induced death rate	0.003 286	Fitted
$\delta_2$	Clinically ill human disease-induced death rate	0.055 487	Fitted
γ	Clinically ill rate	0.500000	Fitted
, )	Recovery rate of Clinically ill humans	0.036 246	Fitted
J	Natural recovery rate due to immunity	0.088 366	Fitted
9	Progression rate from exposed rodents to infected rodents	0.032 386	Fitted
<i>x</i> <sub>1</sub>	Contact rate between infected humans and susceptible humans	0.022 325	Fitted
χ <sub>2</sub>	Contact rate between infected rats and susceptible human	0.052 466	Fitted
<i>т</i>	Contact rate between infected rodents and susceptible rodents	0.012 458	Fitted

population of human is  $N_h = \frac{\phi_h}{\mu_h}$ . The total population of Nigeria is 214,028,300 [35], the recruitment rate is obtained as 64 850 *per weeks*. The remaining parameters are fitted based on the reported cumulative cases of infected humans from January to December 2019. This information was taken from the Nigerian Centre for Disease Control (NCDC) database [8]. The nonlinear least square technique was used to fit the model using the mathematical program MATLAB-R2017b. Table 2 shows all of the parameter values that were estimated and fitted, and figure 2 shows the data fitting of the observed cumulative cases.

#### 4. Fractional calculus

Fractional calculus has a wide range of applications in numerous branches of science, including mathematical biology [21, 37–40]. The fractional framework for the monkeypox model in (1) is given as follows:

$$\begin{cases} {}_{0}^{CF} D_{t}^{\wp} S_{h} = \phi_{h} - (\lambda_{h} + \mu_{h}) S_{h} \\ {}_{0}^{CF} D_{t}^{\wp} E_{h} = \lambda_{h} S_{h} - (\mu_{h} + \beta) E_{h} \\ {}_{0}^{CF} D_{t}^{\wp} I_{h} = \beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1}) I_{h} \\ {}_{0}^{CF} D_{t}^{\wp} C_{h} = \gamma I_{h} - (\rho + \mu_{h} + \delta_{2}) C_{h} \\ {}_{0}^{CF} D_{t}^{\wp} R_{h} = \rho C_{h} + \omega I_{h} - \mu_{h} R_{h} \\ {}_{0}^{CF} D_{t}^{\wp} S_{r} = \phi_{r} - \lambda_{r} S_{r} - \mu_{r} S_{r} \\ {}_{0}^{CF} D_{t}^{\wp} E_{r} = \lambda_{r} S_{r} - (\epsilon + \mu_{r}) E_{r} \\ {}_{0}^{CF} D_{t}^{\wp} I_{r} = \epsilon E_{r} - \mu_{r} I_{R} \end{cases}$$

$$(24)$$

where  ${}_{0}^{CF}D_{t}^{\wp}$  represents Caputo-Fabrizio derivative with fractional order  $\wp$ . In the upcoming sub-section, we will introduce the results of fractional-calculus for investigation of system (24) of monkeypox.

#### 4.1. Fractional-calculus results

For the analysis of our monkeypox dynamics, we will now explain the fundamental concepts and findings of Caputo-Fabrizio (CF) fractional derivatives. The following definitions apply to these essential ideas:

**Definition 1.** The CF fractional derivative [41] for a function  $h \in H^1(a, b)$  is as follows

$$D_t^{\wp}(g(t)) = \frac{\mathcal{U}(\wp)}{1-\wp} \int_a^t g'(x) \exp\left[-\wp \frac{t-x}{1-\wp}\right] dx,$$
(25)

where b > a and  $\mathcal{U}(\tau)$  represents the normality [41] with  $\wp \in [0, 1]$ . In the case, if  $h \notin H^1(a, b)$ , then,

$$D_t^{\wp}(g(t)) = \frac{\wp U(\wp)}{1-\wp} \int_a^t (g(t) - g(x)) \exp\left[-\wp \frac{t-x}{1-\wp}\right] dx.$$
(26)

**Remark 1.** If  $\alpha = \frac{1-\wp}{\wp} \in [0, \infty)$  and  $\wp = \frac{1}{1+\alpha} \in [0, 1]$ , then equation (26) implies



$$D_t^{\wp}(g(t)) = \frac{M(\alpha)}{\alpha} \int_a^t g'(x) e^{[-\frac{t-x}{\alpha}]} dx, \ M(0) = M(\infty) = 1.$$
(27)

Furthermore, we have

$$\alpha \xrightarrow{\lim} 0 \frac{1}{\alpha} \exp\left[-\frac{t-x}{\alpha}\right] = \delta(x-t).$$
(28)

Definition 2. [42], For a given function g, the integral in the fractional framework is expressed as follows

$$I_t^{\wp}(g(t)) = \frac{2(1-\wp)}{(2-\wp)U(\wp)}g(t) + \frac{2\wp}{(2-\wp)U(\wp)}\int_0^t g(u)du, \quad t \ge 0.$$
(29)

where  $\wp$  indicates the fractional order of the integral with  $0 < \wp < 1$ .

Remark 2. The above definition 2 gives the following

$$\frac{2(1-\wp)}{(2-\wp)\mathcal{U}(\wp)} + \frac{2\wp}{(2-\wp)\mathcal{U}(\wp)} = 1,$$
(30)

which implies that  $\mathcal{U}(\wp) = \frac{2}{2-\wp}, 0 < \wp < 1$ . The researchers in [42], introduced the following with help of (30)

$$D_t^{\wp}(g(t)) = \frac{1}{1-\wp} \int_0^t g'(x) \exp\left[\wp \frac{t-x}{1-\wp}\right] dx,$$
(31)

in which the term  $\wp$  is the fractional order with  $0 < \wp < 1$ .

# 5. Analysis of the solutions

Here, we focused on the analysis of the solutions of the hypothesized fractional system of monkeypox disease. Fixed point theory will be applied to investigate the existence of the solution (24). We proceed as follows

$$\begin{cases} S_{h}(t) - S_{h}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \phi_{h} - (\lambda_{h} + \mu_{h}) S_{h} \}, \\ E_{h}(t) - E_{h}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \lambda_{h} S_{h} - (\mu_{h} + \beta) E_{h} \}, \\ I_{h}(t) - I_{h}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1}) I_{h} \}, \\ C_{h}(t) - C_{h}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \gamma I_{h} - (\rho + \mu_{h} + \delta_{2}) C_{h} \}, \\ R_{h}(t) - R_{t}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \rho C_{h} + \omega I_{h} - \mu_{h} R_{h} \}, \\ S_{r}(t) - S_{r}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \phi_{r} - \lambda_{r} S_{r} - \mu_{r} S_{r} \}, \\ E_{r}(t) - E_{r}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \lambda_{r} S_{r} - (\epsilon + \mu_{r}) E_{r} \}, \\ I_{r}(t) - I_{r}(0) = {}_{0}^{CF} I_{t}^{\wp} \{ \epsilon E_{r} - \mu_{r} I_{R} \} \end{cases}$$
(32)

We used the concepts of the research [42] and obtained the following

$$\begin{split} S_{h}(t) - S_{h}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\phi_{h} - (\lambda_{h} + \mu_{h})S_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\phi_{h} - (\lambda_{h} + \mu_{h})S_{h}\}dy, \\ E_{h}(t) - E_{h}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\lambda_{h}S_{h} - (\mu_{h} + \beta)E_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\lambda_{h}S_{h} - (\mu_{h} + \beta)E_{h}\}dy, \\ I_{h}(t) - I_{h}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1})I_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1})I_{h}\}dy, \\ C_{h}(t) - C_{h}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\gamma I_{h} - (\rho + \mu_{h} + \delta_{2})C_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\gamma I_{h} - (\rho + \mu_{h} + \delta_{2})C_{h}\}dy, \\ R_{h}(t) - R_{h}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\rho C_{h} + \omega I_{h} - \mu_{h}R_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\rho C_{h} + \omega I_{h} - \mu_{h}R_{h}\}dy, \\ S_{r}(t) - S_{r}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\phi_{r} - \lambda_{r}S_{r} - \mu_{r}S_{r}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\lambda_{r}S_{r} - (\epsilon + \mu_{r})E_{r}\}dy, \\ E_{r}(t) - E_{r}(0) &= \frac{2(1-\wp)}{(2-\wp)U(\wp)} \{\epsilon E_{r} - \mu_{r}I_{h}\} \\ &+ \frac{2\wp}{(2-\wp)U(\wp)} \int_{0}^{t} \{\epsilon E_{r} - \mu_{r}I_{h}\}dy. \end{split}$$
(33)

Further, we have

$$\begin{cases}
L_{1}(t, S_{h}) = \phi_{h} - (\lambda_{h} + \mu_{h})S_{h}, \\
L_{2}(t, E_{h}) = \lambda_{h}S_{h} - (\mu_{h} + \beta)E_{h}, \\
L_{3}(t, I_{h}) = \beta E_{h} - (\omega + \gamma + \mu_{h} + \delta_{1})I_{h}, \\
L_{4}(t, C_{h}) = \gamma I_{h} - (\rho + \mu_{h} + \delta_{2})C_{h}, \\
L_{5}(t, R_{h}) = \rho C_{h} + \omega I_{h} - \mu_{h}R_{h}, \\
L_{6}(t, S_{r}) = \phi_{r} - \lambda_{r}S_{r} - \mu_{r}S_{r}, \\
L_{7}(t, S_{\nu}) = \lambda_{r}S_{r} - (\epsilon + \mu_{r})E_{r}, \\
L_{8}(t, E_{\nu}) = \epsilon E_{r} - \mu_{r}I_{R}.
\end{cases}$$
(34)

**Theorem 3.** The kernels  $L_1$ ,  $L_2$ ,  $L_3$ ,  $L_4$ ,  $L_5$ ,  $L_6$ ,  $L_7$  and  $L_8$  fulfills the condition of Lipschitz and contraction if the below satisfies

$$0 \leqslant rac{lpha_1 M}{N_h} + rac{lpha_2 I_r}{N_r} + \mu_h < 1.$$

**Proof.** For the above demanded outcomes, we take  $S_h$  and  $S_{h1}$ , and start from  $L_1$  in the following manner

(39)

$$L_{1}(t, S_{h}) - L_{1}(t, S_{h1}) = -\frac{\alpha_{1}I_{h}}{N_{h}} \{S_{h}(t) - S_{h}(t_{1})\} - \frac{\alpha_{2}I_{r}}{N_{r}} \{S_{h}(t) - S_{h}(t_{1})\} - \mu_{h} \{S_{h}(t) - S_{h}(t_{1})\}.$$
(35)

After simplification of (35), we attain the below

$$\begin{aligned} \|L_{1}(t, S_{h}) - L_{1}(t, S_{h1})\| &\leq \|\frac{\alpha_{1}I_{h}}{N_{h}} \{S_{h}(t) - S_{h}(t_{1})\}\| + \|\frac{\alpha_{2}I_{r}}{N_{r}} \{S_{h}(t) - S_{h}(t_{1})\}\| \\ &+ \|\mu_{h} \{S_{h}(t) - S_{h}(t_{1})\}\| \\ &\leq \frac{\alpha_{1}I_{h}}{N_{h}} \|\{S_{h}(t) - S_{h}(t_{1})\}\| + \frac{\alpha_{2}I_{r}}{N_{r}} \|\{S_{h}(t) - S_{h}(t_{1})\}\| \\ &+ \|\mu_{h} \{S_{h}(t) - S_{h}(t_{1})\}\| \\ &\leq \frac{\alpha_{1}M}{N_{h}} \|\{S_{h}(t) - S_{h}(t_{1})\}\| + \frac{\alpha_{2}M}{N_{r}} \|\{S_{h}(t) - S_{h}(t_{1})\}\| \\ &+ \|\mu_{h} \{S_{h}(t) - S_{h}(t_{1})\}\| \\ &\leq \left(\frac{\alpha_{1}M}{N_{h}} + \frac{\alpha_{2}I_{r}}{N_{r}} + \mu_{h}\right) \|S_{h}(t) - S_{h}(t_{1})\}\|. \end{aligned}$$
(36)

Taking  $\mu_1 = \left(\frac{\alpha_1 M}{N_h} + \frac{\alpha_2 I_r}{N_r} + \mu_h\right)$ , where  $I_h \leq M$  and  $I_r \leq M$  due to boundedness, we get the below

$$\|L_1(t, S_h) - L_1(t, S_{h1})\| \leq \mu_1 \|S_h(t) - S_h(t_1)\|.$$
(37)

Thus, we proved the Lipschitz condition for  $L_1$ , in addition to this, the contraction is also obtained from the condition  $0 \leq \left(\frac{\alpha_1 M}{N_h} + \frac{\alpha_2 I_r}{N_r} + \mu_h\right) < 1$ . In the same way, we can determine the Lipschitz conditions as

$$\begin{split} \|L_{2}(t, E_{h}) - L_{2}(t, E_{h1})\| &\leq \mu_{2} \|E_{h}(t) - E_{h}(t_{1})\|, \\ \|L_{3}(t, I_{h}) - L_{3}(t, I_{h1})\| &\leq \mu_{3} \|I_{h}(t) - I_{h}(t_{1})\|, \\ \|L_{4}(t, C_{h}) - L_{4}(t, C_{h1})\| &\leq \mu_{4} \|C_{h}(t) - C_{h}(t_{1})\|, \\ \|L_{5}(t, R_{h}) - L_{5}(t, R_{h1})\| &\leq \mu_{2} \|R_{h}(t) - R_{h}(t_{1})\|, \\ \|L_{6}(t, S_{r}) - L_{3}(t, S_{r1})\| &\leq \mu_{3} \|S_{r}(t) - S_{r}(t_{1})\|, \\ \|L_{7}(t, E_{r}) - L_{7}(t, E_{r1})\| &\leq \mu_{4} \|E_{r}(t) - E_{r}(t_{1})\|, \\ \|L_{8}(t, I_{r}) - L_{5}(t, I_{r1})\| &\leq \mu_{5} \|I_{r}(t) - I_{r}(t_{1})\|. \end{split}$$
(38)

The equation (33) implies the following after simplification

$$\begin{cases} S_{h}(t) = S_{h}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{1}(t,S_{h}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{1}(y,S_{h}))dy, \\ E_{h}(t) = E_{h}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{2}(t,E_{h}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{2}(y,E_{h}))dy, \\ I_{h}(t) = I_{h}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{3}(t,I_{h}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{3}(y,I_{h}))dy, \\ C_{h}(t) = C_{h}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{4}(t,C_{h}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{4}(y,C_{h}))dy, \\ R_{h}(t) = R_{h}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{5}(t,R_{h}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{5}(y,R_{h}))dy, \\ S_{r}(t) = S_{r}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{6}(t,S_{r}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{6}(y,S_{r}))dy, \\ E_{r}(t) = E_{r}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{7}(t,E_{r}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{7}(y,E_{r}))dy, \\ I_{r}(t) = I_{r}(0) + \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)}L_{8}(t,I_{r}) + \frac{2\varphi}{(2-\varphi)U(\varphi)}\int_{0}^{t}(L_{8}(y,I_{r}))dy. \end{cases}$$

(40)

(41)

further, we get

$$\begin{split} S_{hn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_1(t, S_{h(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_1(y, S_{h(n-1)})) dy, \\ E_{hn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_2(t, E_{h(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_2(y, E_{h(n-1)})) dy, \\ I_{hn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_3(t, I_{h(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_3(y, I_{h(n-1)})) dy, \\ C_{hn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_4(t, C_{h(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_4(y, C_{h(n-1)})) dy, \\ R_{hn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_5(t, R_{h(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_r(y, R_{h(n-1)})) dy, \\ S_{rn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_6(t, S_{r(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_6(y, S_{r(n-1)})) dy, \\ E_{rn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_7(t, E_{r(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_7(y, E_{r(n-1)})) dy, \\ I_{rn}(t) &= 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} L_8(t, I_{r(n-1)}) + 2 \frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_8(y, I_{r(n-1)})) dy, \end{split}$$

with the below mentioned initial values

$$S_h^0(t) = S_h(0), E_h^0(t) = E_h(0), I_h^0(t) = I_h(0), C_h^0(t) = C_h(0), R_h^0(t) = R_h(0),$$
  
$$S_r^0(t) = S_r(0), E_h^0(t) = E_r(0), I_r^0(t) = I_r(0).$$

The difference terms are obtained as follows

$$\begin{split} \kappa_{1n}(t) &= S_{hn}(t) - S_{h(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_1(t, S_{h(n-1)}) - L_1(t, S_{h(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_1(y, S_{h(n-1)}) - L_1(y, S_{h(n-2)})) dy, \\ \kappa_{2n}(t) &= E_{hn}(t) - E_{h(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_2(t, E_{h(n-1)}) - L_2(t, E_{h(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_2(y, E_{h(n-1)}) - L_2(y, E_{h(n-2)})) dy, \\ \kappa_{3n}(t) &= I_{hn}(t) - I_{h(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_3(t, I_{h(n-1)}) - L_3(t, I_{h(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_3(y, I_{h(n-1)}) - L_3(y, I_{h(n-2)})) dy, \\ \kappa_{4n}(t) &= C_{hn}(t) - C_{h(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_4(t, C_{h(n-1)}) - L_4(t, C_{h(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_4(y, C_{h(n-1)}) - L_4(y, C_{h(n-2)})) dy, \\ \kappa_{5n}(t) &= R_{hn}(t) - R_{h(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_5(t, R_{h(n-1)}) - L_5(t, R_{h(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_5(y, R_{h(n-1)}) - L_5(y, C_{h(n-2)})) dy, \\ \kappa_{6n}(t) &= S_{rn}(t) - S_{r(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_6(t, S_{r(n-1)}) - L_6(t, S_{r(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_6(y, S_{r(n-1)}) - L_6(y, S_{r(n-2)})) dy, \\ \kappa_{7n}(t) &= E_{rn}(t) - E_{r(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_7(t, E_{r(n-1)}) - L_7(t, E_{r(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_7(y, E_{r(n-1)}) - L_7(y, E_{r(n-2)})) dy, \\ \kappa_{8n}(t) &= I_{rn}(t) - I_{r(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_8(t, I_{r(n-1)}) - L_8(t, I_{r(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_7(y, E_{r(n-1)}) - L_7(y, E_{r(n-2)})) dy, \\ \kappa_{8n}(t) &= I_{rn}(t) - I_{r(n-1)}(t) = \frac{2(1-\varphi)}{(2-\varphi)U(\varphi)} (L_8(t, I_{r(n-1)}) - L_8(t, I_{r(n-2)})) \\ &+ 2\frac{\varphi}{(2-\varphi)U(\varphi)} \int_0^t (L_7(y, E_{r(n-1)}) - L_8(y, I_{r(n-2)})) dy. \end{aligned}$$

Observing that

$$\begin{cases} S_{hn}(t) = \sum_{i=1}^{n} \kappa_{1i}(t), & E_{hn}(t) = \sum_{i=1}^{n} \kappa_{2i}(t), \\ I_{hn}(t) = \sum_{i=1}^{n} \kappa_{3i}(t), & C_{hn}(t) = \sum_{i=1}^{n} \kappa_{4i}(t), \\ R_{hn}(t) = \sum_{i=1}^{n} \kappa_{5i}(t), & S_{rn}(t) = \sum_{i=1}^{n} \kappa_{6i}(t), \\ E_{rn}(t) = \sum_{i=1}^{n} \kappa_{7i}(t), & I_{rn}(t) = \sum_{i=1}^{n} \kappa_{8i}(t). \end{cases}$$
(42)

Evaluating on the same way, we get

$$\|\kappa_{1n}(t)\| = \|S_{hn}(t) - S_{h(n-1)}(t)\| = \left\| 2\frac{(1-\wp)}{(2-\wp)U(\wp)}(L_1(t, S_{h(n-1)}) - L_1(t, S_{h(n-2)})) + 2\frac{\wp}{(2-\wp)U(\wp)} \int_0^t (L_1(y, S_{h(n-1)}) - L_1(y, S_{h(n-2)}))dy \right\|.$$
(43)

The equation (43) implies that

$$\begin{aligned} \|S_{hn}(t) - S_{h(n-1)}(t)\| &\leq 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} \|(L_1(t, S_{h(n-1)}) - L_1(t, S_{h(n-2)}))\| \\ &+ 2 \frac{\wp}{(2-\wp)U(\wp)} \left\| \int_0^t (L_1(y, S_{h(n-1)}) - L_1(y, S_{h(n-2)})) dy \right\|, \end{aligned}$$
(44)

the above leads to

$$\begin{aligned} \|S_{hn}(t) - S_{h(n-1)}(t)\| &\leq 2 \frac{(1-\wp)}{(2-\wp)U(\wp)} \mu_1 \|S_{h(n-1)} - S_{h(n-2)}\| + 2 \frac{\wp}{(2-\wp)U(\wp)} \mu_1 \\ &\times \int_0^t \|S_{h(n-1)} - S_{h(n-2)}\| dy. \end{aligned}$$
(45)

Furthermore

$$\|\kappa_{1n}(t)\| \leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_1\|\kappa_{1(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_1\int_0^t\|\kappa_{1(n-1)}(y)\|dy.$$
(46)

In the similar passion, we have

$$\begin{aligned} \|\kappa_{2n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{2}\|\kappa_{2(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{2}\int_{0}^{t}\|\kappa_{2(n-1)}(y)\|dy, \\ \|\kappa_{3n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{3}\|\kappa_{3(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{1}\int_{0}^{t}\|\kappa_{3(n-1)}(y)\|dy, \\ \|\kappa_{4n}(t)\| &\leq \frac{2(1-\wp)}{(2-\wp)U(\wp)}\mu_{4}\|\kappa_{4(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{4}\int_{0}^{t}\|\kappa_{4(n-1)}(y)\|dy, \\ \|\kappa_{5n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{5}\|\kappa_{5(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{5}\int_{0}^{t}\|\kappa_{5(n-1)}(y)\|dy, \\ \|\kappa_{6n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{6}\|\kappa_{6(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{6}\int_{0}^{t}\|\kappa_{6(n-1)}(y)\|dy, \\ \|\kappa_{7n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{7}\|\kappa_{7(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{7}\int_{0}^{t}\|\kappa_{7(n-1)}(y)\|dy, \\ \|\kappa_{8n}(t)\| &\leq 2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_{8}\|\kappa_{8(n-1)}(t)\| + 2\frac{\wp}{(2-\wp)U(\wp)}\mu_{8}\int_{0}^{t}\|\kappa_{8(n-1)}(y)\|dy. \end{aligned}$$

**Theorem 4.** If one can search a t<sub>0</sub> in a manner that the following condition satisfies

$$2\frac{(1-\wp)}{(2-\wp)U(\wp)}\mu_1+2\frac{\wp}{(2-\wp)U(\wp)}\mu_1t_0<1,$$

then, we have an exact coupled-solutions of the proposed fractional system (24).

**Proof.** As the Lipschitz condition is fulfilled and  $S_h(t)$ ,  $E_h(t)$ ,  $I_h(t)$ ,  $R_h(t)$ ,  $S_r(t)$ ,  $E_r(t)$  and  $I_r(t)$  are bounded. Then, from (46) and (47), we have the following

$$\begin{aligned} \|\kappa_{1n}(t)\| &\leq \|S_{hn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{1} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{1}t \right) \right]^{n}, \\ \|\kappa_{2n}(t)\| &\leq \|E_{hn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{2} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{2}t \right) \right]^{n}, \\ \|\kappa_{3n}(t)\| &\leq \|I_{hn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{3} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{4}t \right) \right]^{n}, \\ \|\kappa_{4n}(t)\| &\leq \|C_{hn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{4} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{4}t \right) \right]^{n}, \\ \|\kappa_{5n}(t)\| &\leq \|R_{hn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{5} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{5}t \right) \right]^{n}, \\ \|\kappa_{6n}(t)\| &\leq \|S_{rn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{6} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{7}t \right) \right]^{n}, \\ \|\kappa_{8n}(t)\| &\leq \|I_{rn}(0)\| \left[ \left( 2\frac{(1-\varphi)}{(2-\varphi)U(\varphi)}\mu_{8} \right) + \left( 2\frac{\varphi}{(2-\varphi)U(\varphi)}\mu_{8}t \right) \right]^{n}. \end{aligned}$$

As a result of this, continuity and existence of the solutions are achieved. Furthermore, we have to show that the above is a solution of our system (24), we proceed as follows

$$S_{h}(t) - S_{h}(0) = S_{hn}(t) - W1_{n}(t),$$

$$E_{h}(t) - E_{h}(0) = E_{hn}(t) - W2_{n}(t),$$

$$I_{h}(t) - I_{h}(0) = I_{hn}(t) - W3_{n}(t),$$

$$C_{h}(t) - C_{h}(0) = C_{hn}(t) - W4_{n}(t),$$

$$R_{h}(t) - R_{h}(0) = R_{hn}(t) - W5_{n}(t),$$

$$S_{r}(t) - S_{r}(0) = S_{rn}(t) - W6_{n}(t),$$

$$E_{r}(t) - E_{r}(0) = E_{rn}(t) - W7_{n}(t),$$

$$I_{r}(t) - I_{r}(0) = I_{rn}(t) - W8_{n}(t).$$
(49)

In the next step, we take

$$\begin{split} \|B_{n}(t)\| &= \left\| \frac{2(1-\wp)}{(2-\wp)U(\wp)} (L_{1}(t,S_{hn}) - L_{1}(t,S_{h(n-1)})) + \frac{2\wp}{(2-\wp)U(\wp)} \right. \\ & \times \int_{0}^{t} (L_{1}(y,S_{hn}) - L_{1}(y,S_{h(n-1)})) dy \, \left\|, \\ & \leqslant \frac{2(1-\wp)}{(2-\wp)U(\wp)} \| (L_{1}(t,S_{hn}) - (L_{1}(t,S_{h(n-1)}))\| + \frac{2\wp}{(2-\wp)U(\wp)} \\ & \times \int_{0}^{t} \| (L_{1}(y,S_{h}) - L_{1}(y,S_{h(n-1)})) \| dy, \\ & \leqslant \frac{2(1-\wp)}{(2-\wp)U(\wp)} \mu_{1} \| S_{h} - S_{h(n-1)} \| + \frac{2\wp}{(2-\wp)U(\wp)} \mu_{1} \| S_{h} - S_{h(n-1)} \| t. \end{split}$$
(50)

Further, we have

$$\|W1_{n}(t)\| \leq \left(\frac{2(1-\wp)}{(2-\wp)U(\wp)} + \frac{2\wp}{(2-\wp)U(\wp)}t\right)^{n+1}\mu_{1}^{n+1}a.$$
(51)

At time  $t_0$ , we have

$$\|W1_{n}(t)\| \leq \left(\frac{2(1-\wp)}{(2-\wp)U(\wp)} + \frac{2\wp}{(2-\wp)U(\wp)}t_{0}\right)^{n+1}\mu_{1}^{n+1}a.$$
(52)

Following the same steps and using (52), we have

$$||W1_n(t)|| \longrightarrow 0, \quad n \to \infty.$$

In a similar passion, we obtain that  $W2_n(t)$ ,  $W3_n(t)$ ,  $W4_n(t)$ ,  $W5_n(t)$ ,  $W6_n(t)$ ,  $W7_n(t)$ ,  $W8_n(t)$  approaches to 0 as *n* approaches  $\infty$ .



- g--

To prove the solution uniqueness of the system (24), we assume

$$(S_{h1}(t), E_{h1}(t), I_{h1}(t), C_{h1}(t), R_{h1}(t), S_{r1}(t), E_{r1}(t), I_{h1}(t))$$

is another solution on contrary manner, then

$$S_{h}(t) - S_{h1}(t) = \frac{2(1 - \wp)}{(2 - \wp)U(\wp)} (L_{1}(t, S_{h}) - L_{1}(t, S_{h1})) + \frac{2\wp}{(2 - \wp)U(\wp)} \times \int_{0}^{t} (L_{1}(y, S_{h}) - L_{1}(y, S_{h1})) \, dy.$$
(53)



By using norm on (53), we have

$$||S_{h}(t) - S_{h1}(t)|| \leq \frac{2(1 - \wp)}{(2 - \wp)U(\wp)} ||L_{1}(t, S_{h}) - L_{1}(t, S_{h1})|| + \frac{2\ell}{(2 - \ell)U(\ell)} \times \int_{0}^{t} ||L_{1}(y, S_{h}) - L_{1}(y, S_{1h})||dy.$$
(54)



Here, we have the following through Lipschitz condition

$$\|S_{h}(t) - S_{h1}(t)\| \leq \frac{2(1-\wp)}{(2-\wp)U(\wp)}\mu_{1}\|S_{h}(t) - S_{h1}(t)\| + \frac{2\wp}{(2-\wp)U(\wp)} \times \int_{0}^{t} \mu_{1}t\|S_{h}(t) - S_{h1}(t)\|dy.$$
(55)



This implies that

$$\|S_{h}(t) - S_{h1}(t)\| \left(1 - \frac{2(1-\wp)}{(2-\wp)U(\wp)}\mu_{1} - \frac{2\wp}{(2-\ell)U(\wp)}\mu_{1}t\right) \leq 0.$$
(56)

Theorem 5. If the following condition satisfied

$$\left(1 - \frac{2(1 - \wp)}{(2 - \wp)U(\wp)}\mu_1 - \frac{2\wp}{(2 - \wp)U(\wp)}\mu_1 t\right) > 0.$$
(57)

then the system (24) has unique solution.



**Proof.** Let us assume that the above (57) satisfies then (56) gives us the following

$$\|S_h(t) - S_{h1}(t)\| = 0, (58)$$

as a result of this, we have

$$S_h(t) = S_{h1}(t).$$
 (59)

In the similar way, we attain the below

$$E_h(t) = E_{h1}(t), I_h(t) = I_{h1}(t),$$
  
 $C_h(t) = C_{h1}(t), R_h(t) = R_{h1}(t),$ 



gure 8. Illustration of the graphical view of the solution pathways of our system with the variation of input factor 
$$\sigma$$
.

 $S_r(t) = S_{r1}(t), E_r(t) = E_{r1}(t),$ 

$$I_r(t) = I_{h1}(t).$$

In the next step, we perform some simulations to examine the transmission process of the proposed fractional system of monkeypox disease. In our simulations, we used the estimated and fitted parameter values as presented in table 2. We performed several simulations to interrogate the importance of parameters on the outcomes of the system of monkeypox disease. In figures 3 and 4, we presented the graphical view analysis of the hypothesized fractional system of monkeypox with different values of fractional order. We observed a significant decrease in the solution pathways of infected individuals of both species and predict that this parameter can be

used as a control parameter. In the second scenario presented in figures 5 and 6, we varied the input parameter  $\omega$ and  $\rho$  respectively to observe the variation in the system dynamics. We noticed the contribution of these parameters in the transmission pathways of infected individuals. In last scenario presented in figures 7 and 8, we noticed the variation in the transmission process of monkeypox with the variation of  $\alpha_2$  and  $\sigma$ . We discovered that contact rates are also important and potentially dangerous in terms of increasing the level of monkeypox infection. Therefore, we recommend these factors to the policymakers for the control of the monkeypox. Based on our results, we predicted that the memory index or fractional order can also be used as a control parameter.

# 6. Conclusion

In this study, a new mathematical model based on the fractional differential system was presented to investigate the transmission dynamics of the monkeypox virus. The two main groups in the population were humans and rodents. The developed model was parameterized using cumulative reported data in Nigeria using NCDC data. The results show that the proposed model fits the data well and can be used to make reliable predictions about the progression of this disease in Nigeria. We show that the model's disease-free is locally asymptotically stable if the threshold quantity  $R_0 < 1$  and unstable otherwise. Through numerical simulations with different input parameters, the system's dynamical behaviour has been demonstrated. On the dynamics of the monkeypox disease, the effects of various input parameters have been considered.

The study's findings are expected to provide a critical paradigm for evaluating monkeypox management. Finally, the current study may serve as a basic guideline for the public health sector in terms of how to effectively manage the country's monkeypox outbreak, which has been described as a major health risk and thus a serious cause for concern.

#### Data availability statement

The data that support the findings of this study are available upon reasonable request from the authors.

#### **ORCID** iDs

Olumuyiwa James Peter D https://orcid.org/0000-0001-9448-1164

#### References

- Centers for Disease Control and Prevention 2021 National Center for Emerging and Zoonotic Infectious Diseases (NCEZID), Division of High-Consequence Pathogens and Pathology (DHCPP), Monkeypox https://www.cdc.gov/poxvirus/monkeypox/index.html accessed: November 10, 2021
- [2] Bunge E M, Hoet B, Chen L, Lienert F, Weidenthaler H, Baer L R and Steffen R 2022 The changing epidemiology of human monkeypoxa potential threat? a systematic review *PLoS Negl. Trop. Dis.* **16** e0010141
- [3] Durski K N, McCollum A M, Nakazawa Y, Petersen B W, Reynolds M G, Briand S, Djingarey M H, Olson V, Damon I K and Khalakdina A 2018 Emergence of monkeypoxwest and central africa, 1970-2017 Morbidity and Mortality Weekly Report 67 306
- [4] Jezek Z, Szczeniowski M, Paluku K, Mutombo M and Grab B 1988 Human monkeypox: confusion with chickenpox Acta Trop. 45 297–307
- [5] Alakunle E, Moens U, Nchinda G and Okeke M I 2020 Monkeypox virus in nigeria: infection biology, epidemiology, and evolution Viruses 12 1257
- [6] Nguyen P-Y, Ajisegiri W S, Costantino V, Chughtai A A and MacIntyre C R 2021 Reemergence of human monkeypox and declining population immunity in the context of urbanization, Nigeria, 2017-2020 Emerg Infect Dis. 27 1007–14
- [7] 2021 Nigeria Centre for Disease Control, Monkeypox outbreak situation report, accessed: November 10, 2021 https://ncdc.gov.ng/ report/
- [8] 2021 Centers for Disease Control and Prevention, National Center for Emerging and Zoonotic Infectious Diseases (NCEZID), Division of High-Consequence Pathogens and Pathology (DHCPP), Monkeypox, accessed: November 11, 2021 https://cdc.gov/poxvirus/ monkeypox/index.html
- [9] Peter O J, Kumar S, Kumari N, Oguntolu F A, Oshinubi K and Musa R 2021 Transmission dynamics of monkeypox virus: a mathematical modelling approach *Modeling Earth Systems and Environment* 1–12
- [10] Bhunu C and Mushayabasa S 2011 Modelling the transmission dynamics of pox-like infections *IAENG International Journal* **41** 1–9
- [11] Peter O J, Viriyapong R, Oguntolu F A, Yosyingyong P, Edogbanya H O and Ajisope M O 2020 Stability and optimal control analysis of an scir epidemic model J. Math. Comput. Sci. 10 2722–53
- [12] Peter O J, Qureshi S, Yusuf A, Al-Shomrani M and Idowu A A 2021 A new mathematical model of covid-19 using real data from pakistan Results in Physics 24 104098
- [13] Ojo M, Gbadamosi B, Olukayode A and Oluwaseun O R 2018 Sensitivity analysis of dengue model with saturated incidence rate Open Access Library Journal 5 1
- [14] Abioye A, Ibrahim M, Peter O, Amadiegwu S and Oguntolu F 2018 Differential Transform Method for Solving Mathematical Model of Seir and Sei Spread of Malaria 40 197–219

- [15] Abioye A I, Peter O J, Ogunseye H A, Oguntolu F A, Oshinubi K, Ibrahim A A and Khan I 2021 Mathematical model of covid-19 in nigeria with optimal control Results in Physics 28 104598
- [16] Ayoola T A, Edogbanya H O, Peter O J, Oguntolu F A, Oshinubi K and Olaosebikan M L 2021 Modelling and optimal control analysis of typhoid fever Journal of Mathematical and Computational Science 11 6666–82
- [17] Peter O J, Ibrahim M O, Oguntolu F A, Akinduko O B and Akinyemi S T 2018 Direct and indirect transmission dynamics of typhoid fever model by differential transform method ATBU, Journal of Science, Technology & Education (JOSTE 6 167–77
- [18] Bankuru S V, Kossol S, Hou W, Mahmoudi P, Rychtář J and Taylor D 2020 A game-theoretic model of monkeypox to assess vaccination strategies *PeerJ* 8 e9272
- [19] Usman S et al 2017 Modeling the transmission dynamics of the monkeypox virus infection with treatment and vaccination interventions Journal of Applied Mathematics and Physics 5 2335
- [20] Peter O J, Yusuf A, Oshinubi K, Oguntolu F A, Lawal J O, Abioye A I and Ayoola T A 2021 Fractional order of pneumococcal pneumonia infection model with caputo fabrizio operator *Results in Physics* 29 104581
- [21] Qureshi S and Jan R 2021 Modeling of measles epidemic with optimized fractional order under caputo differential operator Chaos, Solitons Fractals 145 110766
- [22] Peter O J 2020 Transmission dynamics of fractional order brucellosis model using caputo-fabrizio operator International Journal of Differential Equations 2020 2791380
- [23] Du M, Wang Z and Hu H 2013 Measuring memory with the order of fractional derivative Sci. Rep. 31-3
- [24] Peter O J, Shaikh A S, Ibrahim M O, Nisar K S, Baleanu D, Khan I and Abioye A I 2021 Analysis and dynamics of fractional order mathematical model of covid-19 in nigeria using atangana-baleanu operator Computers Materials, & Continua 66 1823–48
- [25] Oud M A A, Ali A, Alrabaiah H, Ullah S, Khan M A and Islam S 2021 A fractional order mathematical model for covid-19 dynamics with quarantine, isolation, and environmental viral load Advances in Difference Equations 2021 1–19
- [26] Baba I A and Nasidi B A 2021 Fractional order epidemic model for the dynamics of novel covid-19 Alexandria Engineering Journal 60 537–48
- [27] Yao S-W et al 2022 Fractional order covid-19 model with transmission rout infected through environment AIMS Mathematics 7 5156–74
- [28] Chinnathambi R, Rihan F A and Alsakaji H J 2021 A fractional-order model with time delay for tuberculosis with endogenous reactivation and exogenous reinfections Math. Methods Appl. Sci. 44 8011–25
- [29] Aslam M, Murtaza R, Abdeljawad T, ur Rahman G, Khan A, Khan H and Gulzar H 2021 A fractional order hiv/aids epidemic model with mittag-leffler kernel Advances in Difference Equations 2021 1–15
- [30] Kumar D, Singh J, Al Qurashi M and Baleanu D 2019 A new fractional sirs-si malaria disease model with application of vaccines, antimalarial drugs, and spraying Advances in Difference Equations 2019 1–19
- [31] Arafa A, Rida S and Khalil M 2012 Solutions of fractional order model of childhooddiseases with constant vaccination strategy Mathematical Sciences LettersAn International Journal 1 17–23
- [32] Lia Y, Haq F, Shah K, Shahzad M and Rahman G u 2017 Numerical analysis of fractional order pine wilt disease model with bilinear incident rate *Journal of Mathematics and Computer Science* 17 420–8
- [33] Peter O J, Yusuf A, Ojo M M, Kumar S, Kumari N and Oguntolu F A 2022 A mathematical model analysis of meningitis with treatment and vaccination in fractional derivatives *International Journal of Applied and Computational Mathematics* 8 1–28
- [34] Nwasuka S, Nwachukwu I and Nwachukwu P 2019 Mathematical model of the transmission dynamics of lassa fever with separation of infected individual and treatment as control measures *Journal of Advances in Mathematics and Computer Science* 32 1–15
- [35] Central Intelligence Agency, The world factbook, Available from: https://www.cia.gov/the-world-factbook/countries/nigeria/
- [36] Bhunu C, Garira W and Magombedze G 2009 Mathematical analysis of a two strain hiv/aids model with antiretroviral treatment Acta Biotheoretica 57 361–81
- [37] Jan R, Khan M A, Kumam P and Thounthong P 2019 Modeling the transmission of dengue infection through fractional derivatives Chaos, Solitons Fractals 127 189–216
- [38] Jan R et al 2020 A new model of dengue fever in terms of fractional derivative Mathematical Biosciences and Engineering 17 5267-88
- [39] Tang T-Q, Shah Z, Jan R, Deebani W and Shutaywi M 2021 A robust study to conceptualize the interactions of cd4 + t-cells and human immunodeficiency virus via fractional-calculus Phys. Scr. 96 125231
- [40] Srivastava H, Jan R, Jan A, Deebani W and Shutaywi M 2021 Fractional-calculus analysis of the transmission dynamics of the dengue infection, Chaos: An Interdisciplinary *Journal of Nonlinear Science* 31 053130
- [41] Caputo M and Fabrizio M 2015 A new definition of fractional derivative without singular kernel Progr. Fract. Differ. Appl. 11–13
- [42] Losada J and Nieto J J 2015 Properties of a new fractional derivative without singular kernel Progr. Fract. Differ. Appl. 1 87–92