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Modeling and analysis of monkeypox disease using fractional derivatives

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ABSTRACT

The frequency of monkeypox outbreaks and the extent of the projected outbreaks in human populations have both steadily increased. This paper proposes Atangana-Baleanu fractional-order derivatives define in Caputo sense to investigate the kinetics of Monkeypox transmission in Ghana. We determine the stability of the recommended model’s equilibrium points and basic reproduction number. The solution’s existence and originality, as well as the model’s Hyers-Ullam stability, are proven. The models basic reproduction number was found to be $R_0 = 0.1940$. The numerical simulation showed the fractional operator had an influence on the various compartments of the model. The dynamics of the disease in the community were shown to be influenced by fractional-order derivatives, and infections were eradicated within the first five (5) days when $\pi = 0.2$.

1. Introduction

The first human case of the zoonotic orthopox DNA virus, known as monkeypox virus, was reported in the Democratic Republic of the Congo in 1970 [15]. The monkeypox virus, a member of the orthopoxvirus genus in the Poxviridae family, is the culprit behind monkeypox. Three additional human viruses are members of this genus: the vaccinia virus, the cowpox virus, and the smallpox-causing variola virus. Smallpox and monkeypox have similar clinical manifestations, with monkeypox’s early-stage lymphadenopathy serving as a defining characteristic. There have never been any reports of smallpox and monkeypox epidemics spreading into human populations as a result of encounters with animal species in coexisting. Monkeypox is a zoonotic disease that spreads from an unexplained animal reservoir to human populations, whereas smallpox is known to only affect humans. Monkeypox is occasionally introduced into human populations as a result of encounters with animal species in the woods of western and central Africa, particularly in the Republic of Congo, the Central African Republic, Nigeria, and the Democratic Republic of the Congo [16,18]. Every monkeypox outbreak was self-contained, with human transmission pathways stopping before epidemics could develop. Monkeypox looks to be taking over as the primary pox in humans after smallpox was eradicated. The chance of an epidemic spreading to another by close contact with lesions, body fluids, infected objects like bedding, and respiratory droplets [19]. There has been an evidence of human-to - human transmission of the virus in the UK [38], and Democratic Republic of Congo [37]. According to a study in ref. [15] 98 percent of the 528 patients with the condition in 16 countries who were studied were gay. On May 24, 2022, a series of monkeypox cases were reported in Ghana [42]. Since then, there has been 84 confirmed cases through human-to-human transmission of the virus and these were identified through contact tracing [20,41–43].

On June 23, 2022, the World Health Organization designated monkeypox as an “emerging risk of moderate public health concern.” More than 65,000 cases of monkeypox virus infection have been reported globally in 106 countries and five geographic zones since September 2022, resulting in 26 deaths. A combination of factors, including waning smallpox immunity, laxing coronavirus disease 2019 (Covid-19) prevention measures, resuming international travel, and sexual interactions associated with large gatherings, may be to blame for the current global outbreak of monkeypox virus infection in humans [15].

There have been few studies on the disease’s transmission in the past [21–25]. However, mathematical models have been used to study the transmission of diseases such as COVID-19 [34–36] and diseases belonging to the family Poxviridae such as smallpox [26–28], chickenpox [29,30], and cowpox [31]. The authors of [26] proposed a mathematical model to examine the effects of case isolation and ring vaccination for epidemic containment and test the capacity of the health system under various scenarios with available interventions in order to estimate the effects of a smallpox attack in Mumbai, India. The authors of [28] created a mathematical model to explain how smallpox spreads...
after being intentionally released into the environment. To examine the chickenpox in Nigeria, Madaki et al. [29] proposed a deterministic SEIR model combining the method of control used by the national chickenpox and leprosy control programs. Using models of ordinary differential equations, Qureshi [30] studied the 2013 chickenpox outbreak among school children in Schenzen, China. In order to find the model with the maximum efficiency rate, three novel models with the Mittag-Leffler type kernel (Atangana-Baleanu in the Caputo sense), the exponentially decaying type kernel (Caputo-Fabrizio), and the power law type kernel (Caputo) were formulated. Somma et al. [22] developed a mathematical model to study the spread of monkeypox among rodents and human populations. According to the concept proposed in Ref. [22], the human population is provided with a quarantine class and a public awareness campaign to prevent the spread of the disease. Peter et al., [24] created a model was put forth by Grant et al. [25] to investigate how the epidemic spread within Zambia. Kalezhi et al. [34] used a number of mathematical models to understand viral infections [1,2]. Veeresha and Prakasha [33] employed the homotopy analysis method to deal with the problem of the monkeypox virus between humans and rodents in Nigeria. In this study, we propose Atangana-Baleanu fractional-order derivatives defined in Caputo sense are used to describe the model. The population is partitioned into six (6) compartments: Susceptible (S), exposed (E), infected (I), hospitalized (H), recovered (R), and immune individuals (V). The total population is given as N(t) = S(t) + E(t) + I(t) + H(t) + R(t) + V(t).

The rate of recruitment into the susceptible class is \( \gamma \), while the rate of natural death is \( \delta \). The susceptible have a \( \varepsilon \) chance of becoming infected when they get the illness from people in \( H \). The parameters \( \xi_2 \) and \( \xi_3 \) are the recovery rates of an infected person and those hospitalized respectively. The parameters \( \xi_4 \) and \( \xi_5 \) are the disease-induced death rate and infectious rate respectively. The rate of transition from the infected compartment to the hospitalized compartment is given by \( \xi_4 \).

The model is described by the following fractional-order derivative equations.

\[
D^\alpha \xi_1 H_s = \frac{\xi_2 H_s H_e}{N} - \frac{\xi_1 H_s}{\xi^*_1 H_s}, \\
D^\alpha \xi_2 H_e = \frac{\xi_2 H_s H_e}{N} - \frac{\xi^*_2 H_s}{\xi^*_2 + \xi^*_1} H_e, \\
D^\alpha \xi_3 H_A = \xi_2 H_e - \frac{\xi^*_2 H_s + \xi^*_3 + \xi^*_4 + \xi^*_8 \xi_1}{\xi^*_2 + \xi^*_3 + \xi^*_4 + \xi^*_8} H_A, \\
D^\alpha \xi_4 H_0 = \xi_3 H_A - \frac{\xi^*_3 + \xi^*_4 + \xi^*_5}{\xi^*_3 + \xi^*_4 + \xi^*_5} H_0, \\
D^\alpha \xi_5 H_R = \xi_4 H_0 + \xi^*_5 H_1 - \frac{\xi^*_4}{\xi^*_4} H_R, \\
D^\alpha \xi_6 H_V = \xi^*_8 - \xi^*_8 H_V.
\]

With initial conditions \( H_s(0) = H_s0, H_e(0) = H_e0, H_A(0) = H_A0, H_0(0) = H_00, H_R(0) = H_R0, H_V(0) = H_V0 \).

2. Model formulation

By analyzing the model in Ref. [24], we develop a mathematical model that best captures the spread of the virus from person to person, altering [24] to incorporate those who are immune to the infection. Then, Atangana-Baleanu fractional-order derivatives defined in Caputo sense are used to describe the model. The population is partitioned into six (6) compartments: Susceptible \( S \), exposed \( E \), infected \( I \), hospitalized \( H \), recovered \( R \) and immune individuals \( V \). The total population is given as \( N(t) = S(t) + E(t) + I(t) + H(t) + R(t) + V(t) \).

The rate of recruitment into the susceptible class is \( \xi_1 \), while the rate of natural death is \( \xi_2 \). The susceptible have a \( \xi_3 \) chance of becoming infected when they get the illness from people in \( H \). The parameters \( \xi_4 \) and \( \xi_5 \) are the recovery rates of an infected person and those hospitalized respectively. The parameters \( \xi_6 \) and \( \xi_7 \) are the disease-induced death rate and infectious rate respectively. The rate of transition from the infected compartment to the hospitalized compartment is given by \( \xi_8 \).

The flowchart of the model is shown in Fig. 1.

![Flowchart of the monkeypox model.](image-url)
where $G(x) = 1 - x + \frac{x}{(1 + x)^2}$ is the normalized function.

Definition 2.3. The Atangana— Baleanu—Caputo derivative’s pertinent fractional integral is given by the definition [1, 4]

\[
I^\alpha_t h(t) = \frac{(1 - \beta)G(x)}{G(x)^{\frac{1}{\alpha}}} h(t) + \frac{\beta}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - \eta)^{\alpha - 1} h(\eta) d\eta.
\]

They calculated both derivatives’ Laplace transforms and discovered the following:

\[
L\{I^\alpha_t D^\beta_x f(t)\} = \frac{G(x)W(f) - F^\alpha f(0)}{(1 - \beta)(f^\beta + \frac{\beta}{x})},
\]

where $L$ is the Laplace transform operator.

Theorem 2.1. For a function $h \in H[I_1, I_2]$, the following results holds [1, 7]:

\[
\|D^\alpha_x D^\beta_x f(t)\|_2 \leq \frac{\|D^\beta_x f(t)\|_2}{\|D^\alpha_x f(t)\|_2},
\]

where $\|\cdot\|_2$ is the Euclidean norm.

Additionally, the derivatives of Atangana, Baleanu, and Caputo satisfy the Lipschitz criterion [1, 7]:

\[
\|D^\alpha_x D^\beta_x f(t) - D^\alpha_x D^\beta_x f(t)\|_2 \leq \|D^\beta_x f(t)\|_2 \|D^\alpha_x f(t)\|_2.
\]

2.2. Existence and uniqueness of the solutions

This section establishes the existence and distinctiveness of the solutions to system (1).

Using the symbol $X$ to represent a banach space, where $X = [0, b]$ and $Y = X \times X \times X \times X \times X \times X \times X$, and $Z(X) = X \times X \times X \times X \times X \times X \times X$. Using the Banach space, the norm $\|H_b, H_a, H_c, H_d, H_e, H_f\|_2 = \|H_b\|_2 + \|H_a\|_2 + \|H_c\|_2 + \|H_d\|_2 + \|H_e\|_2 + \|H_f\|_2$, where $H_b = \sup_{t \in X} H_b$, $H_a = \sup_{t \in X} H_a$, $H_c = \sup_{t \in X} H_c$, $H_d = \sup_{t \in X} H_d$, $H_e = \sup_{t \in X} H_e$, $H_f = \sup_{t \in X} H_f$, and using the ABC integral operator system (1) gives

\[
\begin{align*}
H_b(t) - H_b(0) &= D^\alpha_x D^\beta_x H_b = \xi^3 - \xi^4 H_b, \\
H_a(t) - H_a(0) &= D^\alpha_x D^\beta_x H_a = \xi^2 - \xi^3 H_a, \\
H_c(t) - H_c(0) &= D^\alpha_x D^\beta_x H_c = \xi^1 H_c, \\
H_d(t) - H_d(0) &= D^\alpha_x D^\beta_x H_d = \xi^3 H_d - \xi^4 H_a, \\
H_e(t) - H_e(0) &= D^\alpha_x D^\beta_x H_e = \xi^3 H_e, \\
H_f(t) - H_f(0) &= D^\alpha_x D^\beta_x H_f = \xi^3 H_f.
\end{align*}
\]

Definition 1 gives

\[
\begin{align*}
H_b(t) - H_b(0) &= \frac{-1}{G(x)} \psi_1(\pi, t, H_b(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_1(\pi, r, H_b(r)) dr, \\
H_a(t) - H_a(0) &= \frac{-1}{G(x)} \psi_2(\pi, t, H_a(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_2(\pi, r, H_a(r)) dr, \\
H_c(t) - H_c(0) &= \frac{-1}{G(x)} \psi_3(\pi, t, H_c(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_3(\pi, r, H_c(r)) dr, \\
H_d(t) - H_d(0) &= \frac{-1}{G(x)} \psi_4(\pi, t, H_d(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_4(\pi, r, H_d(r)) dr, \\
H_e(t) - H_e(0) &= \frac{-1}{G(x)} \psi_5(\pi, t, H_e(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_5(\pi, r, H_e(r)) dr, \\
H_f(t) - H_f(0) &= \frac{-1}{G(x)} \psi_6(\pi, t, H_f(t)) + \frac{\pi}{G(x)^{\frac{1}{\alpha}}} \int_0^t (t - r)^{\alpha - 1} \psi_6(\pi, r, H_f(r)) dr.
\end{align*}
\]

where

\[
\begin{align*}
\psi_1(\pi, r, H_b(t)) &= \xi^3 - \xi^4 H_b, \\
\psi_2(\pi, r, H_a(t)) &= \xi^2 - \xi^3 H_a, \\
\psi_3(\pi, r, H_c(t)) &= \xi^1 H_c, \\
\psi_4(\pi, r, H_d(t)) &= \xi^3 H_d - \xi^4 H_a, \\
\psi_5(\pi, r, H_e(t)) &= \xi^3 H_e, \\
\psi_6(\pi, r, H_f(t)) &= \xi^3 H_f.
\end{align*}
\]

Additionally, the Atangana—Baleanu in Caputo derivatives only meets the Lipschitz requirement [7] if $H_b(t)$, $H_a(t)$, $H_c(t)$, $H_d(t)$, $H_e(t)$, and $H_f(t)$ have an upper bound. Assuming that $H_b(t)$ and $H_a(t)$ are pair functions,

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 = \frac{\|H_b(t) - H_b(t)\|_2}{\xi^3 + \xi^4 H_b}.
\]

Considering

\[
F_1 = \left\|\frac{\xi^3 + \xi^4 H_b}{\xi^3 + \xi^4 H_b}\right\|_2.
\]

Equation (10) simplifies to

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 \leq F_1 \left\|H_b(t) - H_b(t)\right\|_2.
\]

Similarly,

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 \leq F_1 \left\|H_b(t) - H_b(t)\right\|_2.
\]

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 \leq F_1 \left\|H_b(t) - H_b(t)\right\|_2.
\]

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 \leq F_1 \left\|H_b(t) - H_b(t)\right\|_2.
\]

\[
\left\|\psi_1(\pi, t, H_b(t)) - \psi_1(\pi, t, H_b(t))\right\|_2 \leq F_1 \left\|H_b(t) - H_b(t)\right\|_2.
\]

Lipschitz’s condition is thus valid. Now, repeatedly applying system (8) results in
\[ H_{\alpha}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_1(\sigma, r, H_{\alpha-1}(r)) \, dr, \]

\[ H_{\beta}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_2(\sigma, r, H_{\beta-1}(r)) \, dr, \]

\[ H_{\gamma}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_3(\sigma, r, H_{\gamma-1}(r)) \, dr, \]

\[ H_{\delta}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_4(\sigma, r, H_{\delta-1}(r)) \, dr, \]

\[ H_{\vartheta}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_5(\sigma, r, H_{\vartheta-1}(r)) \, dr, \]

\[ H_{\zeta}(t) - H_0(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_6(\sigma, r, H_{\zeta-1}(r)) \, dr, \]

\[ \Psi_{H_\alpha}(t) = \Psi_{H_\alpha}(t) - \Psi_{H_{\alpha-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_1(\sigma, r, H_{\alpha-1}(r)) \, dr, \]

\[ \Psi_{H_\beta}(t) = \Psi_{H_\beta}(t) - \Psi_{H_{\beta-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_2(\sigma, r, H_{\beta-1}(r)) \, dr, \]

\[ \Psi_{H_\gamma}(t) = \Psi_{H_\gamma}(t) - \Psi_{H_{\gamma-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_3(\sigma, r, H_{\gamma-1}(r)) \, dr, \]

\[ \Psi_{H_\delta}(t) = \Psi_{H_\delta}(t) - \Psi_{H_{\delta-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_4(\sigma, r, H_{\delta-1}(r)) \, dr, \]

\[ \Psi_{H_{\vartheta}}(t) = \Psi_{H_{\vartheta}}(t) - \Psi_{H_{\vartheta-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_5(\sigma, r, H_{\vartheta-1}(r)) \, dr, \]

\[ \Psi_{H_{\zeta}}(t) = \Psi_{H_{\zeta}}(t) - \Psi_{H_{\zeta-1}}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_6(\sigma, r, H_{\zeta-1}(r)) \, dr, \]

with the initial conditions \( H_0(0) = H_{\alpha_0}, H_0(0) = H_{\beta_0}, H_0(0) = H_{\gamma_0}, \)

\( H_0(0) = H_{\delta_0}, H_0(0) = H_{\vartheta_0}, H_0(0) = H_{\zeta_0}. \)

Difference of consecutive terms yields

\[
\begin{align*}
\Psi_{\alpha_0}(t) &= H_{\alpha}(t) - H_{\alpha-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_1(\sigma, r, H_{\alpha-1}(r)) \, dr, \\
\Psi_{\beta_0}(t) &= H_{\beta}(t) - H_{\beta-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_2(\sigma, r, H_{\beta-1}(r)) \, dr, \\
\Psi_{\gamma_0}(t) &= H_{\gamma}(t) - H_{\gamma-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_3(\sigma, r, H_{\gamma-1}(r)) \, dr, \\
\Psi_{\delta_0}(t) &= H_{\delta}(t) - H_{\delta-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_4(\sigma, r, H_{\delta-1}(r)) \, dr, \\
\Psi_{\vartheta_0}(t) &= H_{\vartheta}(t) - H_{\vartheta-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_5(\sigma, r, H_{\vartheta-1}(r)) \, dr, \\
\Psi_{\zeta_0}(t) &= H_{\zeta}(t) - H_{\zeta-1}(t) = \frac{1 - \pi}{B(\pi)} \int_0^t (t-r)^{\pi-1} \psi_6(\sigma, r, H_{\zeta-1}(r)) \, dr.
\end{align*}
\]
Theorem 2.2. system (1) has a unique solution for \( t \in [0, b] \) subject to the condition \( \frac{1}{m^2} F_i + \frac{\pi}{m^2} b_i F_i < 1, i = 1, 2, 3, \ldots, n \) holds [44].

Proof:
Since \( H_0(t), H_2(t), H_4(t), H_6(t) \) and \( H_V(t) \) are bounded functions and Equation (12)-(13) holds. In a recurring manner (17) reaches

\[
\|\Psi_{H_0}(t)\| \leq \|H_0(t)\| \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

\[
\|\Psi_{H_2}(t)\| \leq \|H_2(t)\| \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

\[
\|\Psi_{H_4}(t)\| \leq \|H_4(t)\| \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

\[
\|\Psi_{H_6}(t)\| \leq \|H_6(t)\| \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

\[
\|\Psi_{H_V}(t)\| \leq \|H_V(t)\| \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

(18)

and.

\[
\|\Psi_{H_0}(t)\| \rightarrow 0, \|\Psi_{H_2}(t)\| \rightarrow 0, \|\Psi_{H_4}(t)\| \rightarrow 0, \|\Psi_{H_6}(t)\| \rightarrow 0, \|\Psi_{H_V}(t)\| \rightarrow 0.
\]

Incorporating the triangular inequality and for any \( j \), system (18)

\[
H_3(t) - \frac{1 - \pi}{G(\pi)} \Psi_1(\pi, t, H_3(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_1 \int_0^t (t - r)^{-1} \Psi_1(\pi, r, H_3(t)) dr \leq \omega_1,
\]

\[
H_5(t) - \frac{1 - \pi}{G(\pi)} \Psi_2(\pi, t, H_5(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_2 \int_0^t (t - r)^{-1} \Psi_2(\pi, r, H_5(t)) dr \leq \omega_2,
\]

\[
H_7(t) - \frac{1 - \pi}{G(\pi)} \Psi_3(\pi, t, H_7(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_3 \int_0^t (t - r)^{-1} \Psi_3(\pi, r, H_7(t)) dr \leq \omega_3,
\]

\[
H_9(t) - \frac{1 - \pi}{G(\pi)} \Psi_4(\pi, t, H_9(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_4 \int_0^t (t - r)^{-1} \Psi_4(\pi, r, H_9(t)) dr \leq \omega_4,
\]

\[
H_{11}(t) - \frac{1 - \pi}{G(\pi)} \Psi_5(\pi, t, H_{11}(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_5 \int_0^t (t - r)^{-1} \Psi_5(\pi, r, H_{11}(t)) dr \leq \omega_5,
\]

\[
H_{13}(t) - \frac{1 - \pi}{G(\pi)} \Psi_6(\pi, t, H_{13}(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_6 \int_0^t (t - r)^{-1} \Psi_6(\pi, r, H_{13}(t)) dr \leq \omega_6,
\]

yields

\[
\|H_{n+1}(t) - H_n(t)\| \leq \sum_{i=1}^{n+1} F_i \left(1 - \frac{\pi}{B(x)} F_1 + \frac{b}{B(x)} F_2 \right)^n,
\]

(19)

where \( F_i = \frac{1}{\pi} F_1 + \frac{\pi}{m^2} b_i F_1 < 1 \).

Hence there exists unique solution for system (1).

2.3. Hyers–Ulam stability

Definition 2.4. Atangana-Baleanu fractional derivative system (1) is said to be Hyers-Ulam stable if constants \( h_i < 0, i \in N^0 \) matching the following conditions exist for any \( \alpha_i > 0, i \in N^0 \)

Table 1

| Parameter description | Description | Value, Year | Source |
|-----------------------|-------------|-------------|--------|
| \( \xi_1 \) Contact rate between infected human and susceptible human | 0.022325 | 11 |
| \( \xi_2 \) Recruitment rate | 29.08 | 12, 13 |
| \( \xi_3 \) The rate at which critically ill individuals recover | 0.036246 | 11 |
| \( \xi_4 \) The rate at which infected individuals recover due to natural immunity | 0.088366 | 11 |
| \( \xi_5 \) The rate at which infected individuals become critically ill | 0.5 | 11 |
| \( \xi_6 \) The rate at which the exposed becomes infected | 0.016744 | 11 |
| \( \xi_7 \) Monkeypox disease-induced death rate | 0.003286 | 11 |
| \( \xi_8 \) Natural mortality rate | 0.4252912 \times 10^{-4} | 12 |
| \( \eta \) Immunity rate | 0.1 | 11 |

and there exist \( \{H_5(t), H_{13}(t), H_6(t), H_{19}(t), H_{27}(t) \} \), where

\[
\dot{H}_5(t) = \frac{1 - \pi}{G(\pi)} \Psi_1(\pi, t, H_5(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_1 \int_0^t (t - r)^{-1} \Psi_1(\pi, r, H_5(t)) dr,
\]

\[
\dot{H}_7(t) = \frac{1 - \pi}{G(\pi)} \Psi_2(\pi, t, H_7(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_2 \int_0^t (t - r)^{-1} \Psi_2(\pi, r, H_7(t)) dr,
\]

\[
\dot{H}_9(t) = \frac{1 - \pi}{G(\pi)} \Psi_3(\pi, t, H_9(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_3 \int_0^t (t - r)^{-1} \Psi_3(\pi, r, H_9(t)) dr,
\]

\[
\dot{H}_{11}(t) = \frac{1 - \pi}{G(\pi)} \Psi_4(\pi, t, H_{11}(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_4 \int_0^t (t - r)^{-1} \Psi_4(\pi, r, H_{11}(t)) dr,
\]

\[
\dot{H}_{13}(t) = \frac{1 - \pi}{G(\pi)} \Psi_5(\pi, t, H_{13}(t)) + \frac{\pi}{G(\pi)} \frac{1}{\pi} F_5 \int_0^t (t - r)^{-1} \Psi_5(\pi, r, H_{13}(t)) dr,
\]

(21)

such that,

\[
\|H_5(t) - \dot{H}_5(t)\| \leq \mu_1 \omega_1, \quad \|H_7(t) - \dot{H}_7(t)\| \leq \mu_2 \omega_2, \quad \|H_9(t) - \dot{H}_9(t)\| \leq \mu_3 \omega_3,
\]

\[
\|H_{11}(t) - \dot{H}_{11}(t)\| \leq \mu_4 \omega_4, \quad \|H_{13}(t) - \dot{H}_{13}(t)\| \leq \mu_5 \omega_5,
\]

\[
\|H_{15}(t) - \dot{H}_{15}(t)\| \leq \mu_6 \omega_6,
\]

\[
\|H_{17}(t) - \dot{H}_{17}(t)\| \leq \mu_7 \omega_7,
\]

\[
\|H_{19}(t) - \dot{H}_{19}(t)\| \leq \mu_8 \omega_8,
\]

\[
\|H_{21}(t) - \dot{H}_{21}(t)\| \leq \mu_9 \omega_9,
\]

\[
\|H_{23}(t) - \dot{H}_{23}(t)\| \leq \mu_{10} \omega_{10},
\]

\[
\|H_{25}(t) - \dot{H}_{25}(t)\| \leq \mu_{11} \omega_{11},
\]

\[
\|H_{27}(t) - \dot{H}_{27}(t)\| \leq \mu_{12} \omega_{12},
\]
\[ \mu_q \cdot \frac{\partial}{\partial t}, |\bar{H}_q(t) - \dot{\bar{H}}_q(t)| \leq \mu_q \cdot \frac{\partial}{\partial t}, |\bar{H}_u(t) - \dot{\bar{H}}_u(t)| \leq \mu_q \cdot \frac{\partial}{\partial t}, |\bar{H}_v(t) - \dot{\bar{H}}_v(t)| \leq \mu_q \cdot \frac{\partial}{\partial t}. \]

3. Equilibrium point and local stability

3.1. Equilibrium points

We examine the equilibrium points/steady states of the model in this section. The disease-free equilibrium (DFE) and the endemic equilibrium (EE) are the two steady states of system (1). When there is no infection in the population, or when \( H_A = H_Q = 0 \), the steady state solution is disease-free equilibrium. Solving system (1) after equating the right side of the system to zero results in

\[ DFE = (H_e^+, H_e^+, H_e^+, H_e^+, H_e^+, H_e^+) = \left( \frac{\xi^*}{\xi_1}, 0, 0, 0, \frac{\xi^* \eta^*}{\xi_1} \right) \quad (22) \]

The endemic equilibrium point, \( EE = (H_e^+, H_e^+, H_e^+, H_e^+, H_e^+, H_e^+) \) is

\[ \begin{align*}
H_e^+ &= \frac{\xi^* N}{\xi_2 H_A + \xi_1^2 N} H_e^+ \\
H_e^+ &= \frac{\xi^*}{(\xi_2 + \xi_1^2 + \xi_1)} H_e^+ \\
H_e^+ &= \frac{\xi^* \eta^*}{\xi_1} H_e^+ \\
H_e^+ &= \frac{\xi^* \eta^*}{\xi_1} H_e^+ \\
H_e^+ &= \frac{\xi^* \eta^*}{\xi_1} H_e^+ \\
\end{align*} \quad (23) \]

The basic reproductive number is the total number of secondary cases that a single infected person might cause during the course of the infection, in a susceptible population [1]. It is a crucial factor that determines whether or not the disease will spread throughout a population. The infected compartments in this model are \( H_e, H_A \) and \( H_Q \). Denote \( F \) and \( V \), respectively, as the right-hand side of system (1) corresponding to the infected compartments using the next-generation operator method [1].

\[ \frac{d}{dt} = F(x) - V(z) \]

Where,

\[ F = \left( \frac{\xi^* H_A H_e}{N}, \frac{\xi^* H_e H_A}{N}, \frac{\xi^* H_e H_Q}{N} \right) \quad \text{and} \quad V = \left( \frac{(\xi_5^* + \xi_7^*) H_e}{\xi_1}, \frac{(\xi_5^* + \xi_7^*) H_e}{\xi_1}, \frac{(\xi_5^* + \xi_7^*) H_e}{\xi_1} \right) \]

The matrix \( F(x) \) and \( V(x) \) calculated at the equilibrium point of disease-free is given as.

\[ \begin{align*}
F &= \left( \begin{array}{ccc}
0 & \frac{\xi^* H_e^+}{N} & 0 \\
\frac{\xi^* H_e^+}{N} & 0 & 0 \\
0 & \frac{\xi^* H_e^+}{N} & 0
\end{array} \right) \\
V &= \left( \begin{array}{ccc}
\xi_5^* + \xi_7^* & 0 & 0 \\
0 & \xi_5^* + \xi_7^* + \xi_4^* + \xi_8^* & 0 \\
0 & 0 & (\xi_6^* + \xi_8^* + \xi_4^* + \xi_7^*)
\end{array} \right) \\
FV^{-1} &= \left( \begin{array}{ccc}
\frac{\xi_7^*}{\xi_5^* + \xi_7^*} & 0 & 0 \\
0 & \frac{\xi_4^*}{\xi_5^* + \xi_7^*} & 0 \\
0 & \frac{\xi_6^*}{\xi_5^* + \xi_7^*} & 0
\end{array} \right) \\
\end{align*} \]

The basic reproductive number is the largest positive eigenvalue of \( FV^{-1} \) and is given as

\[ R_0 = \frac{\xi^* \eta^*}{(\xi_2 + \xi_4^* + \xi_1^2 + \xi_3^*) (\xi_5^* + \xi_7^*)} \quad (24) \]

Fig. 2. Dynamics of the susceptible compartment.

Fig. 3. Dynamics of the exposed compartment.

3.2. Local stability of the disease-free equilibrium

The following theorem establishes the necessary condition for the local stability of the disease-free steady state.

**Theorem 3.1.** In the event that it exists, the disease-free equilibrium is locally asymptotically unstable for \( R_0 > 1 \) and stable for \( R_0 < 1 \).

The Jacobian matrix of system (1), evaluated at the disease-free equilibrium point is given as

\[ J = \begin{bmatrix}
-\frac{\xi^*}{\xi_1} & 0 & -\frac{\xi^*}{\xi_1} & 0 & 0 & 0 \\
0 & 0 & -\frac{\xi^*}{\xi_1} & 0 & 0 & 0 \\
0 & 0 & -\frac{\xi^*}{\xi_1} & 0 & 0 & 0 \\
0 & 0 & \xi^* & 0 & 0 & -\frac{\xi^*}{\xi_1} \\
0 & 0 & \xi^* & 0 & 0 & -\frac{\xi^*}{\xi_1} \\
0 & 0 & \xi^* & 0 & 0 & -\frac{\xi^*}{\xi_1}
\end{bmatrix} \quad (25) \]

We must demonstrate the negative real components of each and every eigenvalue of system (25). The first four (4) eigenvalues are \( \theta_{1,2,3} \)
= −ξ_{1}, and φ_{4} = −(ξ_{9}^\pi + ξ_{1}^\pi + ξ_{1}^\pi). The remaining ones are derived from the sub-matrix (26), which is created by leaving off the first, fourth, fifth, and sixth rows and columns of system (25). Thus, we have

\[ J_{E} = \begin{pmatrix} -Ι(ξ_{9}^\pi + ξ_{1}^\pi) & ξ_{9}^\pi \\ ξ_{9}^\pi & -Ι(ξ_{9}^\pi + ξ_{1}^\pi + ξ_{1}^\pi + ξ_{1}^\pi) \end{pmatrix}. \] (26)

System (26) characteristic equation, is given as

\[ φ^2 + f_1 φ + f_2 = 0, \] (27)

where

\[ f_1 = ξ_{9}^\pi + ξ_{4}^\pi + ξ_{1}^\pi + 2ξ_{1}^\pi, \]
\[ f_2 = (ξ_{9}^\pi + ξ_{4}^\pi + ξ_{1}^\pi + ξ_{1}^\pi)(ξ_{9}^\pi + ξ_{1}^\pi)(1 - R_0). \]

The stability of the aforementioned characteristic equation is then assessed using the Routh—Hurwitz stability criterion. According to the Routh-Hurwitz stability criterion, if both the \( f_1 > 0 \) and \( f_2 > 0 \) conditions are satisfied, all of the characteristic equation’s roots have negative real portions, indicating a stable equilibrium. When \( R_0 < 1 \), the coefficients \( f_1 > 0 \) and \( f_2 > 0 \) are clearly greater than zero. As a result, our equilibrium is secure.
3.3. Local stability of the endemic equilibrium point

The endemic equilibrium point is a stable, positive situation in which the disease is still present in the populace.

**Theorem 3.2.** The model has a unique endemic equilibrium point if the following Routh–Hurwitz conditions are satisfied: \( g_1 > 0, g_2 > 0 \) and \( g_1 g_2 > g_3 \). [32]

**Proof.** At the endemic equilibrium point (4), the Jacobian matrix is

\[
J_E = \begin{pmatrix}
J_{11} & 0 & J_{13} & 0 & 0 & 0 \\
J_{12} & J_{22} & J_{23} & 0 & 0 & 0 \\
0 & \xi_6 & J_{33} & 0 & 0 & 0 \\
0 & 0 & \xi_5 & J_{44} & 0 & 0 \\
0 & 0 & 0 & \xi_1 & \xi_6 & \xi_5 \\
0 & 0 & 0 & 0 & 0 & \xi_1
\end{pmatrix},
\]

(28)

where

\[
J_{11} = -\left(\frac{\xi_6 H_n}{N} - \xi_1\right), J_{13} = -(\xi_6 + \xi_1 + \xi_5), J_{13} = -\frac{\xi_6 H_n}{N},
\]

\[
J_{22} = \frac{\xi_6 H_n}{N}, J_{23} = \frac{\xi_6 H_n}{N}, J_{44} = -\left(\xi_6 + \xi_1 + \xi_5\right).
\]

The three eigenvalues of the Jacobian matrix (28) are represented by the diagonal elements \( J_{44} \) and \( \xi_1 \), repeated roots. The remaining values are generated by removing the fourth, fifth, and sixth columns and rows of (28). This results in

\[
J_E = \begin{pmatrix}
J_{11} & 0 & J_{13} \\
J_{12} & J_{22} & J_{23} \\
0 & \xi_6 & J_{33}
\end{pmatrix}
\]

(29)

The characteristic equation of Jacobian (29) is given as

\[
\Omega^3 + g_1 \Omega^2 + g_2 \Omega + g_3 = 0
\]

(30)

Where

\[
g_1 = -(J_{13} + J_{11} + J_{12})
\]

\[
g_2 = J_{33}(J_{11} + J_{22}) + J_{11} J_{22} - \xi_6 J_{23}
\]

\[
g_3 = \xi_6(J_{11} J_{22} - J_{12} J_{23}) - J_{11} J_{22} J_{33}
\]

(31)

If the requirements \( g_1 > 0, g_3 > 0 \), and \( g_1 g_2 > g_3 \) are met, then the characteristic equation (30) has negative real roots, which denotes a stable equilibrium according to the Routh—Hurwitz stability criterion.

4. Numerical analysis

This section validates the fractional-order monkeypox model using Ghana’s demographic information, and published parameter values. The parameter values are given in Table 1. When the parameter values given in Table 1 are used, the result of the basic reproduction number is \( R_0 = 0.1940 \), demonstrating that the disease is not endemic in Ghana.

On May 24, 2022, when the first five cases of monkeypox are confirmed by Ghanaian authorities [14], we start our simulation. The population of Ghana is 30.8 million, according to the 2021 Population and Housing Census [12]. The total population under study, \( N = 30.8 \) million, is taken into account in our model, which is in agreement. \( H_0(0) = 30,799,995, H_0(0) = 0, H_q(0) = 5, H_q(0) = 0, H_q(0) = 0, H_q(0) = 0, H_q(0) = 0 \), are the initial conditions we chose. The results of the simulation are displayed in Figs. 2–8, which depicts the dynamic behavior of the susceptible, exposed, infected, hospitalized, deceased, recovered and immune individuals respectively for the period of 100 days.

4.1. Discussion

In Figs. 2–8, are reported, the solutions of system (1) for 5 different values of \( \pi \in [0, 1] \) at step-size 0.2 for a period of 100 days. The decrease in the susceptible population is directly proportional to a reduction in the value of the fractional operator \( \pi \). There is an early peak in the number of exposed and hospitalized individuals as the fractional operator value is reduced (see Figs. 3 and 5). The number of infected individuals is seen to decay within the first 5 days, and this is directly related to the operator value (see Fig. 4). The number of people who die, on the other hand, increases as \( \pi \) decreases (see Fig. 6). The recovered data exhibits an early peak with a fall in the operator value and also shows the crossover effect of the model (see Fig. 7). The immune individuals increases with a decrease in the value of \( \pi \) (see Fig. 8).

5. Conclusion

This work seeks to examine the transmission of the monkeypox disease among humans in a community. Since contacts from human-to-rods are very rare in the Country. This study examines the spread of the monkeypox virus within a society using the fractional-order derivative described in the Atangana-Baleanu in Caputo sense. The new model includes individuals immune to the virus and also report on the dynamics of the deceased which available model fails to do so. We investigated the qualitative characteristics of the model, including its basic reproduction number, equilibrium points, and equilibrium point stability. Along with Hyers-Ulam stability, the solutions’ existence and distinctiveness were demonstrated. Based on the basic reproduction number \( R_0 = 0.1940 \), it was determined that the disease was not endemic. The numerical simulation revealed that, the fractional operator had an impact on the model’s distinct compartments. Classical models (\( \pi = 1.0 \)) exhibited fewer deaths, however, this wasn’t the case as the fractional operator was reduced. The dynamics of the infected compartment were shown to be influenced by fractional-order derivatives, and infections were eradicated within the first five (5) days when \( \pi = 0.2 \). Control measures could also help curb the disease and prevent future occurrences. Hence, control measures are recommended for future studies.

Author statement

The contribution of each author regarding the manuscript “Modeling and Analysis of Monkeypox using Fractional Derivatives” are as follows: **Samuel Okyere**: Conceptualization, Methodology, Formal analysis, Writing - Original Draft, Software, **Joseph Ackora Prah**: Conceptualization, Methodology, Supervision, Proofreading, Revision.

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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. All parameter values are duly cited and referenced.

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